DOCUMENT RESUME

ED 046 019 CG 006 083

AUTHOR	Springer, Ninfa Saturnino
TITLE	Nutrition and Mental Retardation. An Annotated Bibliography, 1964-1970.
INSTITUTION	Michigan Univ., Ann Arbor. Inst. for the Study of Mental Retardation.
PUB DATE	70
NOTE	67p.
AVAILABLE FROM	Institute for the Study of Mental Retardation, University of Michigan, 611 Church Street, Ann Arbor, Michigan 48104. ($1.00 per copy)
EDRS PRICE	EDRS Price MF-$0.65 HC-$3.29
DESCRIPTORS	*Annotated Bibliographies, Health Education, *Mentally Handicapped, *Mental Retardation, *Nutrition, *Nutrition Instruction

ABSTRACT

This annotated bibliography is primarily organized for nutritionists. It presents selected articles published from 1964 to the present. All aspects of nutrition in mental retardation are covered excepting inborn errors of metabolism. Sections are included on: (1) nutrition, birthweight, and mental retardation; (2) nutrition, growth, and mental retardation; (3) malnutrition and its effects on nervous system development; (4) nutrient metabolism and mental retardation; and (5) techniques in feeding and therapeutic nutrition for the mentally retarded. (TL)

ED046019

an annotated bibliography

1964 - 1970 NUTRITION & MENTAL RETARDATION

Ninfa Saturnino Springer

U.S. DEPARTMENT OF HEALTH, EDUCATION & WELFARE
OFFICE OF EDUCATION
THIS DOCUMENT HAS BEEN REPRODUCED EXACTLY AS RECEIVED FROM THE PERSON OR ORGANIZATION ORIGINATING IT. POINTS OF VIEW OR OPINIONS STATED DO NOT NECESSARILY REPRESENT OFFICIAL OFFICE OF EDUCATION POSITION OR POLICY

INSTITUTE FOR THE STUDY OF MENTAL RETARDATION THE UNIVERSITY OF MICHIGAN

ED046019

NUTRITION AND MENTAL RETARDATION

AN ANNOTATED BIBLIOGRAPHY
1964 - 1970

Ninfa Saturnino Springer, Ph.D., Program Director for Nutrition
Institute for the Study of Mental Retardation
The University of Michigan

CG 006 083

For sale by the Institute for the Study of Mental Retardation
The University of Michigan, 611 Church Street, Ann Arbor,
Michigan 48104 - Price one dollar

CONTENTS

(Reference Number)

PREFACE

GENERAL..... 1 - 11

NUTRITION, BIRTHWEIGHT, AND MENTAL RETARDATION..... 12 - 34

NUTRITION, GROWTH, AND MENTAL RETARDATION..... 35 - 55

MALNUTRITION AND ITS EFFECTS ON NERVOUS SYSTEM DEVELOPMENT

NUTRIENT DEFICIENCY

Multiple Deficiencies in Humans..... 56 - 110

Multiple Deficiencies in Animals..... 111 - 168

Vitamin and Mineral Deficiencies

Thiamine..... 169 - 181

Folic Acid and Vitamin B_{12}..... 182 - 199

Other Vitamins..... 200 - 210

Minerals..... 211 - 215

NUTRIENT INTOXICATION..... 216 - 232

NUTRIENT METABOLISM AND MENTAL RETARDATION

CARBOHYDRATES AND PROTEIN..... 233 - 262

VITAMINS, MINERALS, AND WATER..... 263 - 276

NUTRIENT METABOLISM IN DOWN'S SYNDROME..... 277 - 287

TECHNIQUES IN FEEDING AND THERAPEUTIC NUTRITION FOR THE MENTALLY RETARDED..... 288 - 333

PREFACE

This Annotated Bibliography on Nutrition and Mental Retardation has been prepared and organized primarily for nutritionists. It is intended as an introduction for nutritionists specializing in mental retardation, and as a source of research topics. This does not, however, preclude its use by professionals from other disciplines who wish information on the significance of nutrition in the field of mental retardation.

The Annotated Bibliography presents selected articles published from 1964 to the present. The selections cover all aspects of nutrition in mental retardation excepting inborn errors of metabolism. References on inborn errors of metabolism have been excluded from this bibliography since reports on this topic alone would constitute a separate volume.

The first section, GENERAL, contains articles explaining the role of the nutritionist in facilities serving the mentally retarded. In addition, it includes articles dealing with the broad problem of malnutrition. The second section, NUTRITION, BIRTHWEIGHT, AND MENTAL RETARDATION, emphasizes the importance of nutrition in achieving normal weight at birth, and cites reports which implicate prematurity or low birthweight as a possible cause of mental retardation. The section on NUTRITION, GROWTH, AND MENTAL RETARDATION includes reports on the physical growth and other anthropometric measurements of the mentally retarded. The fourth section, MALNUTRITION AND ITS EFFECT ON NERVOUS SYSTEM DEVELOPMENT, offers voluminous references reflecting the growing interest on this topic. Malnutrition may result from a deficiency or an excessive intake of nutrients. The section has, therefore, been divided into NUTRIENT DEFICIENCY and NUTRIENT INTOXICATION. The former is divided into three subheadings. The subheading Multiple Deficiencies in Humans includes studies showing the effects of malnutrition on the mental development of children and also contains reviews of studies dealing both with humans and with animals. On the other hand, Multiple Deficiencies in Animals deals strictly with studies on an experimental basis. Vitamin and Mineral Deficiencies have further been subdivided for specific vitamins whenever the number of references is sufficient to justify a separate section. It incorporates studies on both humans and animals. NUTRIENT INTOXICATION underscores the teratogenic effects of excessive intakes of certain nutrients. The section on NUTRIENT METABOLISM IN MENTAL RETARDATION contains few references on protein and vitamin metabolism. Those on carbohydrate metabolism stress risk factors involved in diabetes and hypoglycemia. VITAMINS, MINERALS AND WATER deals with the problem of electrolyte balance and the possibility of brain damage due to hypernatremia during infancy. A separate subsection on NUTRIENT METABOLISM IN DOWN'S SYNDROME is given because of the number of reports published on this topic. References published prior to 1964 are included in the section TECHNIQUES IN FEEDING AND THERAPEUTIC NUTRITION FOR THE MENTALLY RETARDED due to the growing interest in this topic by nutritionists working in the clinical setting. In-

cluded also are references that make use of nutrient modification of the diet in the treatment of epilepsy and other conditions associated with mental retardation. Also included are articles which indicate successful nutrient supplementation of diets for the mentally retarded.

Grateful acknowledgement is due Barbara Davis and Robert Thompson for their technical assistance and above all to Margaret Joseph for her invaluable editorial skills and perseverance which made possible the completion of this project.

This publication was supported in part by grant number 923 from the Maternal and Child Health Service of the Health Services and Mental Health Administration.

Nina Jaturnein Springer

June, 1970

GENERAL

AMERICAN ACADEMY OF PEDIATRICS, COMMITTEE ON INTERNATIONAL CHILD HEALTH. Malnutrition in the World's Children. Pediatrics, 43 (1):131, 1969.

The article discusses the extent and occurrence of protein-calorie malnutrition in the world's children and makes recommendations for United States assistance in solving this problem. (1)

BERG, A.D. Malnutrition and National Development. Foreign Affairs, 46(1):126, 1967.

The economic costs of the physical and mental retardation caused by malnutrition, and their effects on national economics are discussed. Research and programs for increasing the world's protein supply are described. The need for an interdisciplinary approach to the malnutrition problems is emphasized. (2)

CULLEY, W.J. Nutrition and Mental Retardation. IN: Carter, C.H., ed. Medical Aspects of Mental Retardation. Springfield, Illinois, 88, 1965.

A review of the nutritional aspects of mental retardation: effect of protein, calorie and vitamin deficiencies on mental development; and the nutritional problems of the mentally retarded child. (3)

HILLIE, H.M. and REEVES, M. The Battle Against Mental Retardation: The Dietitian's Share. Hospitals 38:101, 1964.

A description of the role of the dietitian in the infant and maternity care team is given. (4)

JOURNAL OF TROPICAL PEDIATRICS, 14 (3):149, 1968. Nutrition in Maternal and Child Health (With Special Reference to the Western Pacific).

The monograph reviews nutrient deficiencies; infant and maternal feeding practices; and suggested improvements, as they exist in the Western Pacific. A guide for the treatment of malnourished children, plus recipes, based on South Pacific foods, for infants, toddlers, school children and populations in general is included. Suggestions for nutrition education are given. (5)

LELAND, H. The Relationship Between "Intelligence" and Mental Retardation. American Journal of Mental Deficiency, 73:533, 1969.

The author points out the interrelationships between the biomedical and social aspects of an organism; and the importance of a classification system oriented towards treatment of disorders. (6)

NUTRITION REVIEWS, 27:39, 1969. The Economics of Malnutrition.

Discussion is given of economic costs involved in malnutrition in underdeveloped countries. Malnutrition leads to physical and mental impairments which lead to further economic costs. (7)

PHILLIPS, M.G. Nutrition Opportunities in Specialized Health Areas. Journal of the American Dietetic Association, 55(4):348, 1969.

The article focuses on health programs, funded by the Children's Bureau, to meet needs of low-income families. Health Services for Children and Youth Projects are described in addition to programs especially for the mentally retarded and University Affiliated Centers. The role of nutrition within these programs is defined with an explanation of the functions of nutrition personnel; their academic experience qualifications for positions; specialized training programs designed to prepare individuals for such positions; and sources of information regarding established programs. (8)

STEVENS, H.A. "....The Field is Rich...and Ready for Harvest..." American Journal of Mental Deficiency, 70(1):4, 1965.

A broad discussion on mental retardation includes: possible intellectual function of the mentally retarded; biomedical aspects; cultural familial mental retardation; educational and social work problems; manpower needs; and evaluation of programs and services. (9)

TIZARD, J. Cultural Deprivation and Mental Handicap. Nursing Mirror, 128(17):32, 1969.

A brief discussion is presented on the effect of poor social and environmental conditions, including malnutrition, on the developing child. (10)

UMBARGER, B. Role of the Nutritionist or Dietitian in Clinic Services for the Mentally Retarded. Mental Retardation, 3(5):25, 1965.

The role of the nutritionist or dietitian is outlined. Included is information on services performed, data collection, and nutritional counseling. (11)

NUTRITION, BIRTHWEIGHT, AND MENTAL RETARDATION

ABRAMOWICZ, M., and KASS, E.H., Pathogenesis and Prognosis of Prematurity. New England Journal of Medicine, 275(19):1053, 1966.

Although perinatal death, cerebral palsy, mental retardation, and other neurological disorders occur more frequently in infants of low birth weight (<2500 gm), the effect of low birth weight has not been established. Spastic symmetrical cerebral palsy is more common in prematures. Although reports of the association of mental retardation with prematurity have been conflicting, it does appear that a significant number of very underweight infants are mentally retarded and have other abnormalities. (12)

BREWER, T., Human Pregnancy Nutrition: A Clinical View. Obstetrics and Gynecology, 30(4):605, 1967.

The necessity of adequate pregnancy nutrition is discussed and an adequate prenatal diet described. When maternal nutrition is adequate, the fetal nutrition is more likely to be adequate and prematurity rates may be lower. (13)

BRITISH MEDICAL JOURNAL, 2(5406): 401, 1964. Low Birth Weight and Intelligence.

1066 children weighing less than 4 pounds at birth were admitted to 19 premature baby units in Britain between October 1951 and June 1952 and were followed. It was concluded that if a premature baby escapes congenital defects the intellectual development will be normal, especially if birthweight exceeds 3 lbs. (14)

BULLARD, D.M., Jr., GLASER, H.H. HEAGARTY, M.C., and PIVCHIK, E.C. Failure to Thrive in the "Neglected Child". American Journal of Orthopsychiatry, 37(4):680, 1967.

Failure to thrive, of non-organic causes, is discussed. Investigations have noted parental neglect and various forms of maternal deprivation in these children. The need for direct observations of feeding patterns and nutrient intake, and evaluation of the child and parents in the home are discussed. (15)

CHURCHILL, J.A., NEFF, J.W., and CALDWELL, D.F. Birth Weight and Intelligence. Obstetrics and Gynecology, 28(3):245, 1966.

A study to determine the relationship between birth weight, intelligence, and social class. Significant reduction in intelligence occurred in low birth weight group; no effect of social class found. (16)

CHURCHILL, J.A. The Relationship Between Intelligence and Birthweight in Twins. Neurology, 15 (4):341, 1965.

50 sets of twins were compared for the relationship between birthweight and WISC IQ. The lighter members were found to have lower IQ's. (17)

CLIFFORD, S.H. Prevention of Prematurity the Sine Qua Non for Reduction in Mental Retardation and Other Neurologic Disorders. The New England Journal of Medicine, 271(5):243, 1964.

The identification of the high risk patient followed by special care is necessary to prevent premature births and, thus, lower the perinatal mortality rates. It was felt this would also lower the high morbidity and its accompanying mental and physical injuries. Identification of various high risk patients is discussed. (18)

CREERY, R.D.G. The Infant of Low Birthweight. Nursing Mirror, 125 (3):12, 1967.

Although the premature and the "small for dates" infants are similar in that both have low birthweight, their management and prognosis are appreciably different. Recently it has become known that only about two-thirds of low birthweight infants are premature. The rest are "small for dates" due to intra-uterine growth failure. Unless the diagnosis is in question or the clinical condition is severe, the "small for dates" infant does not have to be transferred to a special baby care unit. They usually do not have respiratory and central nervous system problems; however, they do develop hypoglycemia and hypothermia and do warrant special care. Early tube feeding is probably indicated in many premature and dysmature infants. (19)

GORDON, H.H. Some Biological Aspects of Premature Birth. IN: Askin, J., Cooke, R., and Haller, J.A., Jr., eds. A Symposium on the Child. Baltimore: Johns Hopkins Press, 233, 1967.

The use of rigidly scheduled feeding at 3 hour intervals for premature infants is questioned by data on self-regulated feeding for 20 thriving premature infants. (20)

HARMELING, J.D., and JONES, M.B. Birth Weights of High School Dropouts. American Journal of Orthopsychiatry, 38:63, January, 1968.

Study on all Negro population of high schools in Gainesville, Floriaa to determine if any significant difference in birth weight exists between high school drop-outs and their classmates. The Drop-outs had the lowest average birthweight by sex and in total. The Slow Learners were in middle group, the Normals in the highest birth weight category. (21)

HEIMAR, C.B., CUTLER, R., and FREEDMAN, A.M. Neurological Sequelae of Premature Birth. American Journal of Diseases in Children, 108(2):122, 1964.

The incidence of neurological abnormalities was observed in a group of 319 prematurely born children in the birth weight range of 775-2,100 gm. Results indicated the highest incidence of neurological abnormalities was in the lowest birth weight group, that in the weight range under 1,250 gm. (22)

JACOBSON, H.N., REID, D.E. A Pattern of Comprehensive Maternal and Child Care. The New England Journal of Medicine, 271(6):302, 1964.

Improved medical care for impoverished mothers and young children is necessary to lower the prematurity rates and hence the mortality and morbidity rates. A three fold program is outlined to help attain this goal. (23)

KATZ, C.M., and TAYLOR, P.M. The Incidence of Low Birthweight in Children with Severe Mental Retardation. American Journal of Diseases of Children, 114(1):80, 1967.

The incidence of low birthweight among 573 institutionalized severe mentally retarded children was very high and could not be accounted for solely by socioeconomic factors. The cause of mental retardation was unknown for 369 patients who were termed the undifferentiated group. The incidence of birthweight below 5.5 pounds was 25 percent for the total mentally retarded population and the undifferentiated group, as compared to 7 percent for the general population. This study agrees with other studies that showed an association between prematurity and mental retardation. (24)

KING, L.S. Intrauterine Malnutrition. Journal of the American Medical Association, 191(13): 1077, 1965.

A discussion of the effects of intrauterine malnutrition, outlining the physical and mental subnormalities generally found. (25)

LESLIE, L. Prematurity as an Etiologic Factor in Cerebral Dysfunction. Archives of Physical Medicine and Rehabilitation, 47 (11):711, 1966.

In an attempt to understand the significance of prematurity, its incidence is reviewed in both the normal population and those having cerebral dysfunction. Various pre-natal and neonatal factors predisposing the premature infant to disturbances of the central nervous system are discussed; perhaps the most important correlate of prematurity is the neonate's susceptibility to trauma. Other anatomic and physiologic handicaps of the premature infant are difficulties in regulation of body temperature, respiratory immaturity, and susceptibility to infection, any of which could have an effect on the central nervous system. (26)

LIGHT, I.J., BERRY, H.K., and SUTHERLAND, J.H. Amino Aciduria of Prematurity. American Journal of Diseases of Children, 112(3): 229, 1966.

Aromatic amino acid metabolism in infants born at the Cincinnati, Ohio General Hospital between February 2 and July 20, 1965, weighing less than 2268 gm. were studied. Vitamin C supplementation to their diet helped in controlling serum tyrosine levels. (27)

NAYLOR, A., and MYRIANTHOPOULOS, N. The Relation of Ethnic and Selected Socio-Economic Factors to Human Birth Weight. Annals of Human Genetics, 31(1):71, 1967.

Data from the Collaborative Study on Cerebral Palsy, Mental Retardation and Other Neurological and Sensory Disorders of Infancy and Childhood were used to determine relationships of ethnic and selected socio-economic factors to human birth weight. Foreign or rural birth of the mother, presence of the husband in the household, and low density (number of persons per room) have a positive effect on birth weight in all 3 ethnic groups: Negro, Puerto Rican, and Caucasian. (28)

RILEY, R.L., LANDWIRTH, J., KAPLAN, S.A., and GOLLIPP, P.J. Failure to Thrive: An Analysis of 83 Cases. California Medicine, 108 (1):32, 1968.

Children with a diagnosis of failure to thrive from unknown cause were investigated. With 1/3 of them inadequate home environment was a causative factor. Organic disease, low birth weight, congenital anomalies leading to feeding difficulties, and mental retardation were other causes or influences on the failure to thrive. Association between mental retardation and poor growth are discussed. (29)

ROBOR, I.R., OH, W., WU, P.Y.K., METCOFF, J., VAUGHN, M.A. and GABLER, M. The Effects of Early and Late Feeding of Intrauterine Fetally Malnourished Infants. Pediatrics, 42(2):261, 1968.

Observations suggest that early (4 hour) feeding of infants with intrauterine fetal malnourishment and birth weights below 2,040 gm. may enhance glucose homeostasis in early neonatal life and prevent neonatal symptomatic hypoglycemia. (30)

SINCLARI, J.C., and CALDIRON, J.S. Low Birth Weight and Post Natal Physical Development. Developmental Medicine and Child Neurology, 11:314, 1969.

Some newborn infants of low birth weight tend to retain height and weight deficits. The prognosis for growth appears to be poorer for the term infant with low birth weight than for the preterm low birth weight infant. Cellular basis of growth retarding influences are discussed. (31)

SMALLPIECE, V., and DAVIES, P.A. Immediate Feeding of Premature Infants with Undiluted Breast Milk. Lancet, 2(7374):1349, 1964.

A series of 111 infants seen during a period of 17 months in one hospital was used as cases in a study to determine the effects of early feeding in the possible control of hypoglycemia and hyperbilirubinemia. It was concluded that early feeding was justified for the prevention of hypoglycemia, hyperbilirubinemia, and neurological damage. (32)

STRUTHERS, J.N.M., and KEAY, A.J. Early or Late Feeding of Premature Infants. Nursing Times, 61 (47):1577, 1965.

Sixty-five newborn infants weighing less than 4 1/2 pounds were studied for effects of early or late first feedings. There was a higher incidence of signs of respiratory distress syndrome and a higher bilirubin level for late feeders. Vomiting was more frequent for early feeders. No significant difference in mortality was found. It was suggested by this study that early feeding may have some advantage over delayed feeding. (33)

WALKER, J. Pregnancy and Perinatal Association with Mental Subnormality. IN: Meade, J.E., and Parkes, A.S., eds. Genetic and Environmental Factors in Human Ability. New York: Plenum Press, 1966.

Risk pregnancies and perinatal factors are discussed in terms of their association with mental subnormality. In considering prematurity the general health and physique of the mother is more important than the nature of the pregnancy.

Poor environments associated with poor nutrition may lead to stunted growth, poor obstetric performance, and low birth weight. (34)

NUTRITION, GROWTH, AND MENTAL RETARDATION

BAILIT, H.L., and WHELAN, M.A. Some Factors Related to Size and Intelligence In An Institutional Mentally Retarded Population. Journal of Pediatrics, 71(6):897, 1967.

Anthropometric measurement of 77 institutionalized mentally retarded cases, aged 6 to 25 years, showed them to be smaller in every measurement than a control population. Anthropometric measurements included height, head length, biacromial diameter, bi-iliac diameter, weight, sitting height, bicristal diameter, head breadth, bigonial and bizygomatic measurement, chest breadth, and chest depth. Birth weight was significantly related to IQ and length of institutional stay. There was a positive correlation between age at admission and IQ. Those heavier at birth were larger, more intelligent, and had significantly larger anthropmetric measurements in 6 of 11 parameters. (35)

BONGIOVANNI, A.M. Control of Growth and Development. IN: Askin, J.A., Cooke, R.E., and Haller, J.A., Jr., eds. A Symposium on the Child. Baltimore: Johns Hopkins Press, 255, 1967.

Since growth and development can be affected by factors in the external environment such as emotional disturbance and inadequate nutrition as well as by factors in the internal environment such as major disturbances in chemical composition, genetic factors, the central nervous system, pituitary activity, the functioning of the thyroid hormone, and endocrine secretion, an appreciation of the psychopathology of disease can provide increased understanding of the usual control of human growth. (36)

CHEEK, D.B., and COOKE, R.E. Growth and Growth Retardation. Annual Review of Medicine, 15:357, 1964.

A detailed discussion of the subject is presented and basic information concerning cell number, cell size, and body composition is included. Growth retardation information dealing with malnutrition, inadequate oxygenation, central nervous system disturbances and altered mineral metabolism effects are given. (37)

CRAIG, J.O. Growth of Mental Defectives. Developmental Medicine and Child Neurology, 8(1):87, 1966.

The confusion caused by existing systems of classifying mental retardates on physical growth patterns into different diagnostic categories of retardation was discussed. It was suggested that the number of classes for categorizing mental retardates should be reduced to a few well-defined groups. (38)

CRAVIOTO, J., BIRCH, H.J., DeLICARDIE, E.R., and ROSALES, L. The Ecology of Infant Weight Gain in a Pre-Industrial Society. Acta Paediatrica Scandinavica, 56:71, 1967.

The paper reports the results of an ecological longitudinal study of weight gains made during the first 6 months of life by all children born in a representative Central American village in the course of a 19 month period. (39)

ELMER, E., GREGG, G.S., and ELLISON, P. Late Results of the "Failure to Thrive" Syndrome. Clinical Pediatrics, 8(10):584, 1969.

Non-organic failure to thrive is explained as resulting from poor child care. Emotional deprivation influences pituitary functioning to cause growth failure. Studies on 15 children and their families demonstrated "catch-up growth" as a result of therapeutic intervention. Intellectual growth was found to parallel physical development as damage during infancy is not always reversible, and many such children exhibited mental retardation in later life. (40)

FRANKEL, J.J., and LARON, Z. Psychological Aspects of Pituitary Insufficiency in Children and Adolescents with Special Reference to Growth Hormone. Israel Journal of Medical Science, 4:953, 1968.

Thirty-nine children and adolescents, aged 1 to 20 years, with pituitary insufficiency were assessed by means of a battery of neuropsychological tests to determine possible correlates with the state of endocrine deficiency. Results revealed a lower intelligence quotient for the group as a whole, as well as for a variety of clinical subgroups, when compared with the normal population. There was also a distinct deficiency in visuomotor functioning. An attempt is made to explain these findings on the basis of prenatal or postnatal lack of active growth hormone, with resultant infantile hypoglycemia, an accepted cause of mental and motor retardation. (41)

GRAHAM, G.G. Effect of Infantile Malnutrition on Growth. Federation Proceedings, 26(1):139, 1967.

Longitudinal studies of 53 severely malnourished children show that an inadequate diet can have marked effects on subsequent growth, especially growth of the head. In general, the head circumference and height were significantly below the 50th percentile values even after resumption of proper diet. (42)

JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION, 209(11):1712, 1969. Nature, Nurture, and Stature.

An investigation with underdeveloped and emotionally deprived infants demonstrated that improved diet resulted in rapid growth increases and weight gain. Other changes were not made to the physical and emotional environment of these children, therefore, indicating that neglect of proper feeding was the main cause of the under-development. (43)

KERR, G.R., CHAMOVE, A.S., and HARLOW, H.F. Environmental Deprivation: Its Effect on the Growth of Infant Monkeys. Journal of Pediatrics, 75:833, 1969.

Monkeys reared either in partial or total social isolation for periods longer than three months, were compared to control animals. This experiment suggests that social stimulation is not necessary for normal growth. In children with "deprivation dwarfism", inadequate food intake should be regarded as the probable cause of growth failure. (44)

KERR, G.R., ALLEN, J.R., SCHEFFLER, G., and WAISMAN, H.A. Malnutrition Studies in the Rhesus Monkey. I. Effect on Physical Growth. The American Journal of Clinical Nutrition, 23(6):739, 1970.

In an attempt to define the course and sequelae of malnutrition in one primate species, exact degrees of protein--, or total, calorie malnutrition were produced and investigated in infant rhesus monkeys. Data led to the conclusion that children with edema or emaciation may show only subtle signs of malnutrition; correct diagnosis may not be considered in children in whom the quality of ingested protein results in growth failure without the more dramatic clinical evidence of malnutrition. (45)

KOMROWER, G.M. Failure to Thrive. British Medical Journal, 2(5421): 1377, 1964.

Possible causes of developmental retardation are outlined including: insufficiency of diet, inability to eat and digest food properly, impaired absorption, inability to metabolize properly, and the inability to properly utilize nutrients which have been metabolized. Recommended tests and treatments are also outlined. (46)

KRIEGER, I. Head Circumference, Mental Retardation and Growth Failure. Pediatrics, 37(2):384, 1966.

Twenty-three infants with growth failure without organic disease and 28 infants with idiopathic growth failure were evaluated in regard to stature, head circumference, height, age, and mental retardation showing no correlation between head size and mental retardation. (47)

LOWRY, G.H., BACON, G.E., FISHER, S. and KNOLLER, H. Fasting Growth Hormone Levels in Mentally Retarded Children of Short Stature. American Journal of Mental Deficiency, 73(3):474, 1968.

Fasting serum growth hormone assays were obtained on 46 mentally retarded children with short stature. The etiology of the stunted growth associated with mental deficiency remains speculative, but it apparently is not related to decreased growth hormone levels in the fasting state. (48)

MARSHALL, W.A. Growth in Mentally Retarded Children. Developmental Medicine and Child Neurology, 10(3):390, 1968.

Mental retardation is not pathogenic to abnormal growth; rather, small or excessive stature may be due to the same cause as the low intelligence. Some studies have erroneously correlated mental retardation and growth abnormalities by regarding mental retardation as a complete diagnosis and ignoring underlying pathology. Valid comparisons between retarded and normal populations can be made only if the physical growth of both groups is subject to the same genetic and neuro-endocrine handicaps. (49)

MOSIER, H.D., Jr., GROSSMAN, H.J., and DINGMAN, H.F. Physical Growth in Mental Defectives. Pediatrics, 36(3):465, 1965.

Anthropometric studies were done on 2,472 mentally defective resident

patients at Pacific State Hospital. Ten body dimensions were obtained and the presence or absence of secondary sex development was recorded. Tables of means and standard deviations for each measure by sex, IQ, age, and diagnosis were given. (50)

O'CONNELL, E.J., FELDT, R.H., and STICKLER, G.B. Head Circumference, Mental Retardation, and Growth Failure. Pediatrics, 36 (1):62, 1965.

This study supports the use of head circumference measurement in suspecting mental subnormality in children. (51)

POZSONYI, J. and LOBB, J. Growth in Mentally Retarded Children. Journal of Pediatrics, 71(6):865, 1967.

Investigation of the linear and skeletal development of 958 mentally retarded or non-mentally retarded children found that 2 groups of mentally retarded children with no known organic encephalic disorder were equal in growth to children of normal intelligence which indicates that mentally retarded children are not necessarily physically retarded. Stunting occurred most markedly in mongolism, metabolism disorders, and low birth weight groups. (52)

PRYOR, H.B., AND THELANDER, H.E. Growth Deviations in Handicapped Children: An Anthropometric Study. Clinical Pediatrics, 6(8):501, 1967.

Comparison of the anthropometric measurements of 678 handicapped children with those of 12,000 normal children, ages 1 to 15 years, revealed revealed deviations from the normals in all the handicapped cases except those with a mild neurologic deficit. Results indicated that growth is adversely affected most severely in cases with Down's syndrome followed by those with multiple congenital anomalies. Least affected groups were those with cerebral palsy from birth injury and those with severe hypoxia after normal gestation. Those with only neurological deficit showed no deviation from normals. (53)

RUNDLE, A.T., and SYLVESTON, P.E. Endocrinological Aspects of Mental Deficiency: IV. Growth and Development of Young Females. American Journal of Mental Deficiency, 69:635, 1965.

104 physically healthy female mental defectives, ages 7 to 20 years, were examined. Gross body measurements, (standing and sitting height, biacromial diameter, and bi-iliac width), indicated physical underdevelopment of the females, but the annual percentage increase was similar to normal American girls. (54)

TANNER, J.M. Galtonian Eugenics and the Study of Growth: The Relation of Body Size, Intelligence Test Score, and Social Circumstances in Children and Adults. Eugenics Review, 58(3): 122, 1966.

Studies indicate that body size and mental ability increase in small families, in favorable occupational or socioeconomic classes, and in people who migrate either away from home or upwards in social classes. In children of school age there is a correlation between intelligence scores and body size, this continues at a diminished rate to maturity. (55)

MALNUTRITION AND ITS EFFECTS ON NERVOUS SYSTEM DEVELOPMENT

NUTRIENT DEFICIENCY

Multiple Deficiencies in Humans

ADAMS, R.N. Cultural Aspects of Infant Malnutrition and Mental Development. IN: Scrimshaw, N.S. and Gordon, J.E.. eds. Malnutrition, Learning, and Behavior. Cambridge: MIT Press, 465, 1968.

This paper discusses a broad framework, embracing the full adaptive cycle, that studies investigating the relationship between infant malnutrition and mental development should be based on. This results in expanding the scope of the research with a final goal of a better adaptation of man to the world. (56)

BABSON, S.G., KANGAS, J., YOUNG, N. and BRAMHALL, J.L. Growth and Development of Twins of Dissimilar Size at Birth. Pediatrics, 33(3):327, 1964.

Sixteen pairs of dissimilar sized twins at birth, with a median age of 8 1/2 years were investigated. There were highly significant differences in height, head circumferences and weight; significant differences in intelligence and language comprehension and expression; and no significant differences in articulation, oral structures, oral diadochokinesis or auditory acuity. (57)

BARAITSER, M.E. The Effect of Under-Nutrition on Brain-Rhythm Development. South Africa Medical Journal, 43:56, 1969.

Evidence is presented to validate the hypothesis that electroencephalographic rhythms are affected by early malnutrition. (58)

BARNES, R.H. Early Malnutrition and Behavioral Development. Food and Nutrition News, 39(1), 1967.

Studies on malnourished children by Cravioto and by Stock and Smythe are described. The specific contributions of nutrition and those of social stimulation to mental development can not, as yet, be separated. (59)

BARNES, R.H. Malnutrition in Early Life and Mental Development. New York State Journal of Medicine, 65:2816, 1965.

Evidence from animal and human studies shows that in acute stages of malnutrition, behavioral abnormalities and low scores in psychological development tests are evident. Age of onset and type of malnutrition influence recovery. (60)

BEUKERING, J.V. Undernutrition During Infancy and the Effect of Brain Growth and Intellectual Development. South Africa Medical Journal, 41:1179, 1967.

A brief letter questioning the validity of the work of Stock and Smythe in South Africa is provided. (61)

BIRCH, H.G. Field Measurement in Nutrition, Learning, and Behavior. IN: Scrimshaw, N.S. and Gordon, J.E., eds. Malnutrition, Learning, and Behavior. Cambridge: MIT Press, 497, 1968.

Some of the specific questions to be answered regarding malnutrition and mental development are discussed. Quantitative determinations of the frequency and extent to which malnutrition affects learning and behavior; the relative significance of malnutrition among other causative factors; the ages when malnutrition has most effect on mental development; and the mechanisms by which malnutrition exerts its action are included. Tests must be developed to measure, sensitively, behavioral differences, and, yet they must be relatively insensitive to cultural differences between populations. (62)

BOTHA-ANTON, E., BABAYAN, S., and HARFOUCHE, J.K. Intellectual Development Related to Nutrition Status. Journal of Tropical Pediatrics, 14(3):112, 1968.

A group of children, who had been undernourished before 18 months, had significantly lower IQ's than an adequately nourished group at the age level of 4 to 5 years with no evidence supporting an original difference in IQ. (63)

BRITISH MEDICAL JOURNAL, 1(5588): 333, 1968. Nutrition and the Developing Brain.

Studies on infants with brain damage resulting from improper nutrition, both before (chronic) and immediately after (acute) birth are discussed. Neither hypernatremia, nor hypoglycemia are prevalent causes of neonatal brain damage; however, they are only 2 of possible nutritional disturbances. Detection of persistent difficult behavior in infants, proper diagnosis of the malnutrition, and subsequent treatment are required of pediatricians. (64)

BRITISH MEDICAL JOURNAL, 1:651, 1964. Nutrition of the Brain in Infancy.

Discussion of several studies on malnutrition and brain development is presented. It was concluded that it would be very difficult to prove the effects of malnutrition in early infancy, and the ethical considerations of any prospective studies are mentioned. (65)

BROWN, R.E. Decreased Brain Weight in Malnutrition and its Implications. East Africa Medical Journal, 42:584, 1965.

Review of 1100 autopsies of Ugandan children, aged from birth to 15 years. Those with a history of malnutrition showed significant reduction in body and brain weight. Implications of findings are discussed. (66)

BROWN, R.E. Organ Weight in Malnutrition with Special Reference to Brain Weight. Developmental Medicine and Child Neurology, 8(5):512, 1966.

Heart, liver, spleen, brain, and body weights from autopsies of 1,094 Ugandan children up to 15 years of age were investigated. The children were divided into 2 groups on the basis of whether or not they were undernourished. The weights were compared with those from children of comparable age in other countries. (67)

CABAK, V. and NAJDANVIC, R. Effect of Undernutrition in Early Life on Physical and Mental Development. Archives of Disease in Childhood, 40(213):532, 1965.

The records of 36 children who were admitted for malnutrition to the Hospital for Sick Children in Sarajevo, Yugoslavia, between 1951 and 1957, and later discharged, were selected to compare their present body weight, height, and Binet- Simon IQ as adapted by Stevanovic to those for normals. A correlation was found between deficit in admission weight and present IQ. (68)

CANOSA, C.A. Ecological Approach to the Problems of Malnutrition, Learning, and Behavior. IN: Scrimshaw, N.S., and Gordon, J. E., eds. Malnutrition, Learning, and Behavior. Cambridge: MIT Press, 389, 1968.

The paper reviews some of the complexities and the planning aspects of field studies dealing with malnutrition, learning, and behavior. It is based on a prospective long-term study that began in 1964 at the Institute of Nutrition of Central America and Panama with field operations in Guatemala. The main independent variable is the nutritional state of pre-school children with mental development as the dependent variable. The study attempts to improve understanding of the relationships between malnutrition, learning, and behavior. (69)

CHAMPAKAM, S., SRIKANTIA, S.G., and GOPALAN, C. Kwashiorkor and Mental Development. American Journal of Clinical Nutrition, 21:844, 1968.

Study of nineteen children successfully treated for kwashiorkor. Reductions in intelligence and intersensory organization were found, particularly in the younger age group. The retardation was noticeable mainly with regard to perceptual and abstract abilities. (70)

CHANDRA, R.K. Nutrition and Brain Development. Journal of Tropical Pediatrics, 10(2):37, 1964.

The author reviews some studies on nutritional deficits and their effect on intellectual development and stresses the need for more research in this area. (71)

CHURCHILL, J.A., AYERS, M.A., and CALDWELL, D.F. Intelligence of Children Whose Mothers Have Bilary Tract Disease. Journal of the American Medical Association, 201(6):482, 1967.

A prospective, longitudinal study of children born of mothers with bilary tract disease found that their IQ was lower than the IQ of matched controls. Malabsorption of fats, particularly linoleic acid, may interfere with the lipid supply essential for fetal brain development. (72)

COURSIN, D.B. Effects of Undernutrition on Central Nervous System Function. Nutrition Reviews, 23(3):65, 1965.

The literature on the effects of undernutrition on the central nervous system is reviewed, and suggestions for further research are made. (73)

COURSIN, D.B. Undernutrition and Brain Function. Borden's Review of Nutrition Research, 26(1), 1965.

This paper presents a discussion of the extent of undernutrition in developing countries and its effect on mental capacities. Information on normal development of the brain; the spectrum of mental retardation; the clinical symptoms of severe un-

dernutrition; and the biochemical mechanism abnormalities in protein-calorie undernutrition is included. (74)

CRAVIOTO, J., DeLICARDIE, E.R., and VEGA, L. Amino Acid-Protein Malnutrition and Mental Development. IN: Nyhan, W.L., ed. Amino Acid Metabolism and Genetic Variation. New York: McGraw-Hill, 449, 1967.

The relationship between neurointegrative behavior and nutritional status was examined in 2 groups of children (ages 6 to 11 years) of the same ethnic background. Amino acid-protein malnutrition was defined, retrospectively, as a significant diminuation of height. There is evidence of delayed neurointegrative development in malnourished children. (75)

CRAVIOTO, M., BIRCH, J.G. and GAONA, C.E. Early Malnutrition and Auditory - Visual Integration in School-Age Children. Journal of Special Education, 2:75, 1967.

A study of the ability of malnourished and normal groups of children to integrate visual and auditory information. (76)

CRAVIOTO, J. and ROBLES, B. Evolution of Adaptive and Motor Behavior During Rehabilitation from Kwashiorkor. American Journal of Orthopsychiatry, 35(3):449, 1965.

The psychological test performance during rehabilitation of a group of 20 infants and pre-school children suffering from severe protein-calorie malnutrition was studied by the Gesell method as soon as they had been cured of any acute infectious episode and/or electrolyte disturbance, and afterwards at intervals of 2 weeks during their stay in the nutrition ward. As the patients recovered from malnutrition the difference between chronological age and developmental age in the fields of adaptive, motor, language, and personal-socio behavior was found to decrease except in the group of children whose chronological age on admission was below 6 months. (77)

CRAVIOTO, J., and DeLICARDIE, E.R. Intersensory Development of School-Age Children. IN: Scrimshaw, N.S. and Gordon, J.E., eds. Malnutrition, Learning, and Behavior. Cambridge: MIT Press, 252, 1968.

The relationship between neuro-integrative function and malnutrition in a group of children in a Guatamalan village was studied. The shortest children were assumed to have experienced malnutrition as they showed a lag in development of intersensory competence. This was not the case with urban children studied who were assumed not to have suffered malnutrition. The results of an investigation of auditory-visual capacity of children in a Mexican village are described. Height was once again used as a criterion of evidence of malnutrition. The taller group performed better than the shorter. (78)

CRAVIOTO, J., DeLICARDIE, E.R., and BIRCH, H.G. Nutrition, Growth, and Neurointegrative Development: An Experimental and Ecologic Study. Pediatrics, Supplement 2, 38:319, 1966.

A study using height as a measure of pre-school nutrition was conducted on poor rural children. Results showed a positive correlation between height and intersensory integrative ability. The study was re-

peated on a group of upper class children whose heights were unlikely to have been affected by malnutrition. Here differences in height were not related to intersensory integrative competence. (79)

DAIRY COUNCIL DIGEST, 39(3):13, 1968. Malnutrition in Early Life and Subsequent Mental Performance.

Current concepts of brain development are discussed, and studies investigating the relationship of nutrition to human and animal behavior are described. (80)

DAYTON, D.H. Early Malnutrition and Human Development. Children, 16 (6), 1969.

Studies describing the nutritional status of children in various areas are discussed. Studies on mental development and the effects of malnutrition are provided, and information on methods of nutritional assessment is discussed. (81)

EICHENWALD, H.F. and FRY, P.C. Nutrition and Learning. Science, 163:644, 1969.

This is a broad discussion of the physiological, socio-economic, and psychological factors surrounding nutrition and learning. (82)

FRISCH, R.E. Present Status of the Supposition That Malnutrition Causes Permanent Mental Retardation. American Journal of Clinical Nutrition, 23(2):189, 1970.

The author reviews many of the studies dealing with malnutrition and mental retardation, as well as one study investigating the effects of social-environmental factors on mental development. She concludes that the evidence is not yet sufficient to support the supposition that malnourished children will be permanently mentally retarded. (83)

GRAHAM, G.G. Effect of Infantile Malnutrition. Federation Proceedings, 26(1):139, 1967.

A longitudinal investigation was carried out on 53 severely malnourished children. The earlier in life the deficits occur, the more severe and permanent the stunting of growth, particularly growth in head size. The older the infant when deprived, the longer the period of undernourishment he can tolerate with out permanent stunting. (84)

GRIEVE, S., JACOBSON, S., and PROCTOR, N.S. A Nutritional Myelopathy Occurring in the Bantu on the Witwatersrand. Neurology, 17:1205, 1967.

A series of 61 cases of a nutritional myelopathic syndrome is described. Two autopsies are described in detail, and the main pathological feature in both is degeneration of the lateral pyramidal tracts. A low intake of protein, fats, and vitamins, and often excessive alcohol intake, can be implicated in most cases. (85)

GYORGY, P. Protein-Calorie and Vitamin A Malnutrition in Southeast Asia. Federation Proceedings, 27(3):949, 1968.

Dietary analysis and supplementation were studied. Because some deficiency symptoms are irreversible, prevention is of paramount importance and nutrition education is essential. (86)

HUGHES, E.A., STEVENS, L.H., and WILKINSON, A.W. Some Aspects of Starvation in the Newborn Baby. Archives of Disease in Childhood, 39(208):598, 1964.

Nitrogen, potassium and sodium balance studies were made on 2 newborn infants under starvation. Both infants showed remarkable capacity to resist starvation. (87)

MEDICAL WORLD NEWS, 8(11):60, 1967. Better Diet for Brighter Minds?

Human studies in South Africa, Mexico, and Nazi concentration camps indicate that undernutrition before the age of 4 has intellectual and emotional effects; however, the mature brain can be rehabilitated after starvation. Discussion is presented on enriched food products as a method to combat malnourishment. Fish protein concentrates are described. (88)

MEDICAL WORLD NEWS, 9(37):91, 1968. Deadline for Saving Starved Minds.

Studies show that early malnutrition results in impaired learning ability and that the time available for the nutritional rescue of stunted minds is less than 6 months from birth. Cell division in the human brain continues until 6 months of age; therefore, early good nutrition is the key to a normal complement of brain cells. (89)

MONCKEBERG, F. Effect of Early Marasmic Malnutrition on Subsequent Physical and Psychological Development. IN: Scrimshaw, N.S., and Gordon, J.E., eds. Malnutrition, Learning, and Behavior. Cambridge: MIT Press, 269, 1968.

Studies on children indicate that early malnutrition tends to restrict expression of the genetic potential for development, both physical and psychological. The effects of socioeconomic factors are difficult to separate from nutritional influences. (90)

MORAN, R.E. Possible Causes of Mental Retardation in Puerto Rico. Presented at the 90th Annual Convention of the American Association on Mental Deficiency, May 10, 1966, Chicago, Illinois.

Various etiological factors accounting for mental retardation are discussed. Malnutrition as a result of poverty is given a major role. (91)

NAEYE, R.L. Malnutrition: Probable Cause of Fetal Growth Retardation. Archives of Pathology, 79 (3):284, 1965.

The study compares organ structure of prenatally retarded infants with organ structure of children dying of post natal alimentary malnutrition. Results showed that both groups had abnormal amounts of cutaneous fat. While liver and spleen were disproportionately small, brain, pancreas, heart, lungs and kidney were nearest to normal weight. Malnutrition, therefore, may be a major factor in some cases of antenatal growth retardation. Placental abnormalities suggest a cause. (92)

NATURE, 221:808, 1969. The Developing Brain.

The process of myelination seems to be a "once and for all" event, and therefore is a vulnerable period of development; for even mild undernutrition can permanently affect myelin deposition in the brain. (93)

NUTRITION REVIEWS, 26(7):197, 1968. Undernutrition in Children and Subsequent Brain Growth and Intellectual Development.

The article presents a review of the longitudinal study conducted by Stock and Smythe in South Africa. Evidence is presented that severe undernutrition during the first two years of life is associated with a brain size and an intellectual development below average. Further studies are necessary to separate nutrition from other possible causative factors. (94)

PLATT, B.S., and WHEELER, E.F. Protein-Calorie Deficiency and the Central Nervous System. Developmental Medicine and Child Neurology, 9(1):104, 1967.

Poor educational performance and reduced brain weight are two results of malnutrition in childhood. (95)

POLLITT, E. Ecology, Malnutrition, and Mental Development. Psychosomatic Medicine, 31:193, 1969.

The author points out two major difficulties in malnutrition and intelligence studies: obtaining adequate history, and separating nutritional components from biological and social variables. (96)

RAJALAKSHMI, R. The Psychological Status of Under-Privileged Children Reared at Home and in an Orphanage in South India. Indian Journal of Mental Retardation, 1(2):53, 1968.

Children living in an orphanage in South India and children living in their own homes in a small village near the orphanage were tested for psychological performance. The results showed that nutritional improvement alone does not restore normal psychological functioning in children whose social and cultural environment is totally lacking in emotional and psychological stimulation. (97)

RANDAL, J. Hunger: Does it Cause Brain Damage? Think, 32:2, 1966.

Discussion is given of early malnutrition and mental retardation, and the role of amino acids in brain development. (98)

READ, M.S. Malnutrition and Learning. American Education, 5:10, 1969.

Malnutrition and its extent in the United States is described. Animal and human studies investigating the relationship between malnutrition and mental abilities are discussed. The influence of other variables such as parental, social and environmental are postulated. (99)

SACHDEV, K.K., MANCHANDA, S.S., and LAL, H. The Syndrome of Tremors, Mental Regression, and Anemia in Infants and Young Children: A Study of 102 Cases. Indian Pediatrics, 2:239, 1965.

Complex nutritional factors, rather than specific vitamin deficiency, appears to be at the root of this syndrome. (100)

SCOTT, K.E., and USHER, R. Epiphyseal Development in Fetal Malnutrition Syndrome. New England Journal of Medicine, 270:822, 1964.

X-ray films of the knees were taken in 30 malnourished newborn infants and in 18 normally nourished controls of the same gestational age. The results indicated that fetal malnutrition is accompanied by a delay in epiphysial development. (101)

SCRIMSHAW, N.S. Infant Malnutrition and Adult Learning. Saturday Review, 84:64, 1968.

The paper presents a review of the animal and human studies investigating the effects of malnutrition on physical growth, learning, and behavior. (102)

SCRIMSHAW, N.S. and GORDON, J.E. Malnutrition, Learning, and Behavior. Cambridge: MIT Press, 1968.

The book reports the proceedings of an International Conference on Malnutrition, Learning, and Behavior. It was attended by medical, biological, and social scientists of 38 countries. Both animal and human studies investigating malnutrition and physical and mental development are presented, and the consequences of nutritional deficiencies in the young child on later mental functions are discussed. (103)

SCRIMSHAW, N.S. Malnutrition, Learning, and Behavior. American Journal of Clinical Nutrition, 20: 493, 1967.

A general discussion of the interrelationships between malnutrition, learning, and behavior is given. (104)

STOCK, M.B., and SMYTHE, P.M. Undernutrition During Infancy, and Subsequent Brain Growth and Intellectual Development. IN: Scrimshaw, N.S. and Gordon, J.E., eds. Malnutrition, Learning, and Behavior. Cambridge: MIT Press, 278, 1968.

The results of an 11 year study on 20 Cape Coloured children, who were grossly undernourished in infancy, is reported. Evidence indicates that severe undernutrition during the first 2 years of life, when brain growth is most active, results in a permanent reduction of brain size and a restricted intellectual development. (105)

STOCK, M.B., and SMYTHE, P.M. The Effect of Undernutrition During Infancy on Subsequent Brain Growth and Intellectual Development. South Africa Medical Journal, 41:1027, 1967.

Smaller head circumference, abnormal electroencephalographic patterns and impairment of visual perception was found in a group of malnourished African children. (106)

WINICK, M. Malnutrition and Brain Development. The Journal of Pediatrics, 74(5):667, 1969.

A broad survey of studies, dealing with the effects of malnutrition on the developing brain, are described. Both animal and human studies investigating physical and chemical brain growth are shown, as well as studies dealing with the development of brain function. (107)

WINICK, M. and ROSSO, P. Head Circumference and Cellular Growth of the Brain in Normal and Marasmic Children. Journal of Pediatrics, 74:774, 1969.

The relationship between head circumference and cellular growth of the brain is examined in normal and malnourished children in the first year of life. The data support the validity of using changes in head circumference as a measure of postnatal brain growth. (108)

WINICK, M. and ROSSO, P. The Effect of Severe Early Malnutrition on Cellular Growth of the Human Brain. Pediatric Research, 3:181, 1969.

In ten "normal" brains, obtained from well-nourished Chilean children who died accidentally, weight, protein, and DNA and RNA content were all normal when compared with those values derived from similar children in the United States. In nine infants who died of severe malnutrition during the first year of life, there was a proportional reduction in weight, protein, and RNA and DNA content. These data are similar to previous data in animals and demonstrate that in children, severe early malnutrition can result in curtailment of the normal increase in brain cellularity with increase in age. (109)

WITKOP, C.J. Genetics and Nutrition. Federation Proceedings, 26(1):148, 1967.

Similar biochemical findings exist in histidinemia, phenylketonuria, and kwashiorkor. It is suggested that investigations be done to compare the mental retardation resulting from protein malnutrition with the type of mental retardation resulting from histidinemia and phenylketonuria. (110)

BARNES, R.H., MOORE, A.U., REID, I. M. and POND, W.G. Effects of Food Deprivation on Behavioral Patterns. IN: Scrimshaw, N.S., and Gordon, J.E., eds. Malnutrition, Learning, and Behavior. Cambridge: MIT Press, 203, 1968.

The results of animal studies on pigs and rats investigating the effects of food deprivation on behavior are described. Differences were noted between behavioral changes caused by simple food restriction of normal diet, and those caused by a protein deficient diet. There appeared to be no relationship between the extent of behavioral change and the stunting of body size in animals deprived in early life. (111)

BARNES, R.H. Experimental Approaches to the Study of Early Malnutrition and Mental Development. Federation Proceedings, 26(1): 144, 1967.

Certain types of nutritional deprivation imposed early in life are shown to affect learning capacity. (112)

BARNES, R.H., CUNNOLD, S.R. and ZIMMERMAN, R.R. Influence of Nutritional Deprivations in Early Life on Learning Behavior of Rats as Measured by Performance in a Water Maze. Journal of Nutrition, 89:399, 1966.

Learning behavior was studied in rats that were subjected to different forms of nutritional deprivation in early life. Male rats that were deprived both before and after weaning made significantly more errors than the normal controls. The animals that were deprived pre-weaning or post-weaning alone gave intermediate results. No significant differences were found among female rats subjected to the same treatment regimens. (113)

BARNES, R.H., MOORE, A.U., REID, I. M. and POND, W.G. Learning Behavior Following Nutritional Deprivations in Early Life. Journal of the American Dietetic Association, 51(1):34, 1967.

The results of animal studies investigating the relationship of malnutrition and learning behavior are described. Pigs, malnourished in early life, were unable to extinguish a conditioned response, although both the malnourished and controls were able to develop this response at the same rate. Rats, nutritionally deprived during nursing, made significantly more errors than controls in a water maze test. Rats deprived during the post-weaning period as well as during nursing made the most errors. (114)

BARNES, R.H., MOORE, A.U. and POND, W.G. Behavioral Abnormalities in Young Adult Pigs Caused by Malnutrition in Early Life. Journal of Nutrition, 100(2):149, 1970.

Baby pigs were malnourished for a period of 8 weeks by restricting protein or caloric intake with the objective of studying behavioral changes that remained long after nutritional rehabilitation had been achieved. The nutritional condition which caused the greatest change in behavioral development resulted from feeding a diet very low in protein from the third through the eleventh week of life. (115)

BENTON, J.W., MOSER, H.W., DODGE, P. R. and CARR, S. Modification of the Schedule of Myelination in the Rat by Early Nutritional Deprivation. Pediatrics, 38(5): 801, 1966.

Nutritional deprivation during the first 21 days in newborn rats produced a decrease in myelination. Brain weight, total brain lipids, cholesterol, and phospholipids were decreased 80 percent in the deprived group. (116)

BROWN, M.L. and GUTHRIE, H.A. Effect of Severe Undernutrition in Early Life Upon Body and Organ Weights in Adult Rats. Growth, 32:143, 1968.

Male rats subjected to severe undernutrition from two days following birth until 3, 5, 7 and 9 weeks of age were subsequently fed an adequate diet until reaching adult age of 19 weeks. Body weight and weights of liver, kidneys, heart and brain of all deprived groups were significantly lower than controls and weights of animals deprived for 9 weeks were lower than other deprived groups. (117)

CALDWELL, D.F. and CHURCHHILL, J.A. Learning Ability in the Progeny of Rats Administered a Protein Deficient Diet During the Second Half of Gestation. Neurology, 17:95, 1967.

A group of ten pregnant rats were fed a protein deficient diet, with vitamin supplement, during the second half of gestation. Results showed that subjects receiving the protein deficient diet had longer periods of gestation, weighed less at birth and weaning, and had a higher preweaning mortality rate. In learning ability, the control rats were significantly better in both tests. (118)

CHASE, H.P., DORSEY, J. and McKHANN, G.M. The Effect of Malnutrition on the Synthesis of a Myelin Lipid. Pediatrics, 40(4):551, 1967.

The hypothesis that the period of an active myelin formation is both a cricial and a vulnerable time for the developing central nervous system was strengthened when rats subjected to nutritional deprivation from bith to 21 days of age had a decreased synthesis of sulfatide which was not corrected by refeeding. Malnutrition produced a significant decrease of brain weight, brain lipid, and body weight. Refeeding resulted in a rapid gain of body weight but a slower gain of brain weight and lipids. (119)

CHASE, H.P., LINDSLEY, W.F., Jr., and O'BRIEN, D. Undernutrition and Cerebellar Development. Nature, 221:554, 1969.

The purpose of this study is to ascertain whether malnutrition has any regionally specific effect on cell growth. Rats were undernourished by creating nursing litters of 16 animals to one mother. Body and total brain weight were significantly lower in undernourished animals. DNA was significantly lower-- wholly as a result of the difference in cerebellar DNA content. The effects of undernutrition can be interpreted as less protein and smaller cells in the cerebrum, but with no difference in cell numbers. (120)

CHOW, B.F., BLACKWELL, R.Q. and SHERWIN, R.W. Nutrition and Development. Borden Review of Nutrition Research, 29:25, 1968.

The effects on animals of maternal dietary restricitions are described. It was concluded that both fetal and neonatal undernutrition permanently affect the offspring, and that maternal nutrition in the rat influences both the fetal and neonatal nutrition. (121)

COLLIER, G.H. and SQUIBB, R.I. Malnutrition and the Learning Capacity of the Chicken. IN: Scrimshaw, N.S. and Gordon, J.E., eds. Malnutrition, Learning, and Behavior. Cambridge: MIT Press, 236, 1968.

No abnormal motivation or learning responses were found in chickens placed on a protein deficient diet for the first two weeks after hatching, and then re-fed and examined from 6 weeks onward. (122)

COWLEY, J.J. and GRIESEL, R.D. The Effect on Growth and Behavior of Rehabilitating First and Second Generation Low Protein Rats. Animal Behavior, 14:506, 1966.

Two experiments are described in which rats fed on a low protein diet were rehabilitated on a laboratory diet containing a higher percentage of protein. The retardation in the growth and development became more marked as successive generations of rats were reared on the low protein diet. It is concluded that a low protein diet fed to one generation of rats affects the growth and development of the next generation; this seems to be due, at least in part, to a deficiency in the nutrients received from the mother during the foetal period. (123)

COWLEY, J.J. Time, Place and Nutrition: Some Observations from Animal Studies. IN: Scrimshaw, N. S. and Gordon, J.E., eds. Malnutrition, Learning, and Behavior. Cambridge: MIT Press, 218, 1968.

Rats fed on a low protein diet after weaning showed no significant change in problem solving behavior from controls. If rats and their offspring were retained on the diet, the problem solving ability of succeeding generations was increasingly affected. Protein synthesis was stimulated by supplementing these rats with Dianabol. These rats showed some improvement in problem solving and appeared to be "more purposive" than controls. It was also noted that rats, mice and hamsters restrict their food intake when moved from one territory to another. Analogies between these observations and the human situation were made. (124)

CULLEY, W.J. and MERTZ, E.T. Effect of Restricted Food Intake on Growth and Composition of Preweaning Rat Brain. Proceedings of the Society for Experimental Biology and Medicine, 118(1):233, 1965.

The incorporation of non-lipid solids, cholesterol, phospholipids, and cerebrosides into the brain was reduced by restricting food intake of rats from 5 until 20 days of age. (125)

CULLEY, W.J. and LINEBERGER, R.O. Effect of Undernutrition on the Size and Composition of the Rat Brain. Journal of Nutrition, 96 (3):375, 1968.

Studies with rats indicated that the ability of the rat brain to recover from undernutrition is established somewhere between 11 and 17 days of age. The effects of the restricted feeding were greatest in the cerebellum and least in the pons medulla. (126)

DAVISON, A.N. The Influence of Nutritional Disorders on the Lipid Composition of the Central Nervous System. Proceedings of the Nutrition Society, 27(1):83, 1968.

Research investigating the relationship between malnutrition and lipid composition of the brain is discussed. Evidence indicates that permanent effects may be produced only during a vulnerable period of development. More investigations are needed to relate this to intellectual performance. (127)

DAVISON, A.N. and DOBBING, J. Myelination as a Vulnerable Period in Brain Development: The Foetus and the Newborn. British Medical Bulletin, 22(1):40, 1966.

The deposition of myelin as a critical process in the later phase of brain development is discussed. The "vulnerable" period in human myelin development is probably between the seventh intrauterine month to the first few months of postnatal life. (128)

DICKERSON, J.W.T. and DOBBING, J. Prenatal and Postnatal Growth and Development of the Central Nervous System of the Pig. Proceedings of the Royal Society of London, 166(1005):384, 1967.

The growth and maturation of the central nervous system of pigs was studied by quantitative chemical methods. The most rapid period of growth was from about 6 weeks before birth to about 5 weeks afterwards. This study shows that the time when the spinal cord and the brain, or the cerebellum in particular, may be most susceptible to insult such as undernutrition is determinable. (129)

DICKERSON, J.W. and WALMSLEY, A.L. The Effect of Undernutrition and the Subsequent Rehabilitation on the Growth and Composition of the Central Nervous System of the Rat. Brain, 90:897, 1967.

Weanling rats were undernourished until they were 11 weeks old. Some of them were then allowed unlimited access to food for one, two or eight weeks. The effect of undernutrition and subsequent rehabilitation on the weight of the brain, and on the weight and "thickness" of the spinal cord was studied. The brain and the cord were analysed for total N, total P, DNA-P and cholesterol. (130)

DOBBING, J. Effects of Experimental Undernutrition on Development of the Nervous System. IN: Scrimshaw, N.S. and Gordon, J.E., eds. Malnutrition, Learning, and Behavior. Cambridge: MIT Press, 181, 1968.

The effect of undernutrition on the brain lipids of pigs is described. In rehabilitated pigs, the brains are smaller and they seem to be undermyelinated. The total number of cells is permanently reduced. Results of rat experiments support data that the effects of undernutrition on the brain depends on the timing of the undernutrition in relation to the period of fastest brain growth. (131)

DOBBING, J. The Effect of Undernutrition on Myelination in the Central Nervous System. Biologia Neonatorum, 9(1-6):132, 1966.

Three separate animal experiments support the hypothesis that undernutrition, if sufficiently severe and occurring during critical periods, can affect myelination. (132)

DOBBING, J. and WIDDOWSON, E.M. The Effect of Undernutrition and Subsequent Rehabilitation on Myelination of Rat Brain as Measured by its Composition. Brain, 88 (2):357, 1965.

The effect of undernutrition and subsequent unlimited nourishment on myelination of the brains of 200 rats was investigated. The study clearly showed the ability of the adult brain to synthesize large quantities of cholesterol during rehabilitation after a period of undernutrition. (133)

FRANKOVA, S. and BARNES, R.H. Effect of Malnutrition in Early Life on Avoidance Conditioning and Behavior of Adult Rats. Journal of Nutrition, 96(4):485, 1968.

Rats, undernourished both before and after weaning, showed behavioral disturbances but did not differ significantly in learning rates from controls. Animals restricted in the preweaning period only did not show signs of these disturbances. (134)

FRANKOVA, S. and BARNES, R.H. Influence of Malnutrition in Early Life on Exploratory Behavior of Rats. Journal of Nutrition, 96 (4):477, 1968.

Investigations of rats indicated that undernutrition in the first weeks of life may cause a long term decrease of exploratory drive, despite rehabilitation with a satisfactory diet fed ad libitum from 21 days of age. This effect was found to be greater in males than females. (135)

FRANKOVA, S. Nutritional and Psychological Factors in the Development of Spontaneous Behavior in the Rat. IN: Scrimshaw, N.S. and Gordon, J.E., eds. Malnutrition, Learning, and Behavior. Cambridge: MIT Press, 312, 1968.

Investigations on rats to study the effects of undernutrition and the effects of stimulation on the degree of spontaneous activity of the animals are reported. A reduced caloric intake in the early stages of development results in decreased spontaneous activity, but the effect may be modified appreciably by stimulation. (136)

GEISON, R.L. and WAISMAN, H.A. Effects of Nutritional Status on Rat Brain Maturation as Measured by Lipid Composition. Journal of Nutrition, 100(3):315, 1970.

Alteration of the rate of growth of suckling rats produces changes in the concentrations of several brain lipid components (galactolipid, cholesterol, plasmalogens) and chloroform-methanol extractable protein (proteolipid protein). The results suggest that the rates of lipid accumulation in suckling rats are related to the body and brain growth. (137)

GRANOFF, D.M. and HOWARD, E. Malnutrition in Infancy and Brain Development. Journal of Pediatrics 75:732, 1969.

The importance of regional determination of DNA in the brain, and the need to clarify the functional significance of these alterations in brain composition is discussed. (138)

GUTHRIE, H.A. and BROWN, M.L. Effect of Severe Undernutrition in Early Life on Growth, Brain Size and Composition in Adult Rats. Journal of Nutrition, 94:419, 1968.

A study was undertaken to determine the effect of varying periods of undernutrition in postnatal life on the size and chemical composition of the brain in rehabilitated adult animals. Undernutrition during suckling caused a degree of stunting of body size that was only partially reversed by nutritional rehabilitation. Brain size and brain DNA were depressed by undernutrition in the preweaning period but were not affected further by deprivation in the postweaning period. (139)

HOWARD, E. and GRANOFF, D.M. Effect of Neonatal Food Restrictions in Mice on Brain Growth, DNA, and Cholesterol; and on Adult Delayed Response Learning. Journal of Nutrition, 95:111, 1968.

This study was designed to examine the long-term effects of a limited period of nutritional restriction on ultimate brain size and functional capacity. At 9 months, body, cerebral and cerebellar weights were reduced in the males by 17, 7 and 14%, respectively, below control values. Total DNA was reduced 8% in the cerebrum and 22% in the cerebellum. Cerebral cholesterol was reduced slightly. Despite these brain changes, the restricted groups showed no lasting impairment in voluntary running, in learning a Lashley type III maze, or a visual discrimination with escape from water as a reward. (140)

HURLEY, L.S. The Consequences of Fetal Impoverishment. Nutrition Today, 3(4):2, 1968.

The role of nutritional factors on prenatal development is discussed in general. Specific investigation on the offspring of zinc and manganese deficient animals during pregnancy are described. (141)

McCANCE, R.A. Some Effects of Undernutrition. Journal of Pediatrics, 65(6):1008, 1964.

Experiments investigating the effects of undernutrition, as well as undernutrition and rehabilitation, on pigs, guinea pigs and rats are described. (142)

MEDOVY, H. New Parameters in Neonatal Growth--Cell Number and Cell Size. Journal of Pediatrics, 71 (3):459, 1967.

Studies in the rat fetus show that early malnutrition interfered with cell division, and later malnutrition caused a reduction of individual cell size. If the number of cells in the brain is decreased, mental retardation can be anticipated; however, if the cells are only decreased in size, an increase in growth may catch up to normal size. (143)

MILLEN, J.W. and WOOLLAM, D.H.M. Maternal Nutrition in Relation to Abnormal Fetal Development. Proceedings of the Nutrition Society, 19(1):1, 1960.

A review of research investigating the relationship between maternal nutrition and fetal abnormalities is provided. (144)

MOUREK, J., HIMWICH, W.A., MYSLIVECEK, J. and CALLISON, D.H. The Role of Nutrition in the Development of Evoked Cortical Responses in Rats. Brain Research, 6: 241, 1967.

Maturation of evoked cortical responses to visual and auditory stimulation was investigated in normal as well as in acute and chronically starved rats. Some probable mechanisms of these changes are discussed. (145)

NUTRITION REVIEWS, 27:146, 1969. Cellularity of Rat Adipose Tissue in Relation to Growth, Starvation, and Obesity.

There are indications that the cellularity of adipose tissue can be permanently modified only during the early life--i.e. nutritional influences during this time may be extremely important. Certain metabolic processes in adipose tissue seem to be a function of cell number and not cell size. (146)

NUTRITION REVIEWS, 22(8):244, 1964. Diet, Development and Intelligence.

Second generation rats fed a marginal African type diet showed retarded growth and development and poorer intelligence test scores than did control rats. (147)

NUTRITION REVIEWS, 23:211, 1965. Early Weaning, Diet and Intelligence.

Rats weaned at 15 days of age to a stock ration showed a poorer ability to master and to remember a maze than those weaned at 30 days. When rats were weaned at an earlier age to a high fat diet they responded to the maze test as well or better than those weaned at the later date. (148)

NUTRITION REVIEWS, 25:334, 1967. Underfeeding and Brain Development.

This is a review of a study on young rats that were selectively undernourished in total quantity of food, but not in quality. Brain weight was reduced in the deprived rats, and brain total lipid, phospholipid, and cholesterol were reduced by the same relative amounts as brain weight. (149)

NUTRITION REVIEWS, 25:185, 1967. Undernutrition and Development of the Central Nervous System in the Pig.

A review of a study on growth of spinal cord and brain regions in undernourished and control groups of pigs. (150)

OGATA, K., KIDO, H., ABE, S., FURUSAWA, Y. and SATAKE, M. Activity of Protein Synthesis of the Brain of Protein-Deficient Rats. IN: Scrimshaw, N.S. and Gordon, J.E., eds. Malnutrition, Learning, and Behavior. Cambridge: MIT Press, 131, 1968.

The incorporation of C^{14}-leucine into the total protein by gray matter slices and by a cell-free S_{12}- fraction consisting of microsomes and cell sap from adult rat brain cortex was unaffected by deprivation of dietary protein for 7 days. (151)

OLEWINE, D.A., BARROWS, C.H., Jr. and SHOCK, N.W. Effects of Reduced Dietary Intake on Random and Voluntary Activity in Male Rats. Journal of Gerontology, 19(2): 230, 1964.

Weanling rats subjected to 50 percent dietary restriction showed a reduction in total random movements, an increase in voluntary wheel activity and increased activity before presentation of food as compared with controls. These effects were reversible. (152)

PLATT, B.S. and STEWART, R.J. Effects of Protein-Calorie Deficiency on Dogs. 1. Reproduction, Growth and Behavior. Developmental Medicine and Child Neurology, 10:3, 1968.

Dogs maintained from weaning on diets of low protein value grow slowly and develop changes in their bones, brains and behavior. Many of the abnormalities of gait, growth and electroencephalographic pattern disappear when the deficient animals are given diets of high protein value. (153)

PLATT, B.S. and STEWART, R.J. Effects of Protein-Calorie Deficiency in Dogs. 2. Morphological Changes in the Nervous System. Developmental Medicine and Child Neurology, 11:174, 1969.

The morphological changes observed in the dogs are discussed in relation to clinical signs described in malnourished children. It is suggested that malnutrition during intrauterine and early postnatal life may lead to irreversible changes within the central nervous system. (154)

RAJALAKSHMI, R., GOVINDARAJAN, K., and RAMAKRISHNAN, C. Effects of Dietary Protein Content on Visual Discrimination, Learning and Brain Biochemistry in the Albino Rat. Journal of Neurochemistry, 12(4):261, 1965.

Groups of albino rats, aged 1 month, 6 months and 12 months were maintained on protein deficient diets for 4 to 6 months. They were tested psychologically, then killed, and the brain was analyzed chemically. They were found to perform less well on visual discrimination learning than controls, and some enzymes and amino acids levels were low. (155)

SCHAIN, R.J., CARVER, M.J., COPENHAVER, J.H. and UNDERDAHL, N.R. Protein Metabolism in the Developing Brain: Influence of Birth and Gestational Age. Science, 156(3777):984, 1967.

Brain synthesis decreases with age in young animals primarily in the first days of life. Whether this decrease is due to gestational age or to birth itself is unclear. Experiments with newborn pigs suggest that birth factors result in a sharp decrease in amino acid incorporation into brain protein, irrespective of gestational age. (156)

SERENI, F., PRINCIPI, N., PERLETTI, L. and SERENI, L.P. Undernutrition and the Developing Rat Brain. Biologia Neonatorum, 10 (5):254, 1966.

Acetylcholinesterase activity, norepinephrine concentration, and 5-OH-tryptamine concentration in rat brain were significantly lowered by undernutrition during the suckling period. Undernutrition caused a slowing of body weight and brain weight, growth affecting most those

rats subjected to undernutrition for 35 days. (157)

SHERWIN, R.W. Perinatal Nutrition as a Developmental Determinant. Nutrition News, 30(4):13, 1967.

The effects on rats of manipulation of the maternal diet during gestation and/or lactation are discussed. Data showed the inadequacy of body weight as an index of functional development. (158)

SIMONSON, M., SHERWIN, R.W., HANSON, H.H. and CHOW, B.F. Maze Performance of Offspring of Underfed Mother Rats. Federation Proceedings, 27:727, 1968.

Progeny of mother rats on 50% dietary restricition during gestation and lactation were fed ad libitum after weaning. They had means of running time and errors double that of controls during the first trial on an elevated multiple T maze. Thereafter they required more trials to reach a certain criterion and made more errors. (159)

SIMONSON, M., SHERWIN, R.W., ANILANE, J.K., YU, W.Y. and CHOW, B. F. Neuromotor Development in Progeny of Underfed Mother Rats. Journal of Nutrition, 98(1):18, 1969.

Offspring of mother rats, subjected to 50% dietary restrictions during gestation alone or during gestation and lactation, displayed delays of up to two weeks in neuromotor development. Lesser delays occurred with the less dietary restricted rats. Abnormal activity occurred with both types of progeny, but weight deficits did not occur with those restricted during gestation alone. (160)

SIMONSON, J., YU, W., ANILANE, J. K., SHERWIN, R. and CHOW, B.F. Studies of Development in Progeny of Underfed Rats. Federation Proceedings, 26:519, 1967.

Progeny of rats on 50% restricted diets during gestation and lactation demonstrated physical development, initiation of movement and response to stimuli later than controls but at a lower body weight. Movements requiring extensive coordination showed the longest delay and did not occur at a lower body weight. (161)

STEWART, R.J.C. and PLATT, B.S. Nervous System Damage in Experimental Protein-Calorie Deficiency. IN: Scrimshaw, N.S. and Gordon, J.E., eds. Malnutrition, Learning, and Behavior. Cambridge: MIT Press, 168, 1968.

Nervous system changes in protein-calorie deficient pigs are described, as well as nervous system changes in dogs born to protein deprived mothers. (162)

WINICK, M. Food, Time, and Cellular Growth of the Brain. New York Journal of Medicine, 69:302, 1969.

A general restatement of his article in Nutrition Review, July, 1968 is given. (163)

WINICK, M. Nutrition and Cell Growth. Nutrition Review, 26: 195, 1968.

Discussion of the use of DNA content to measure brain cell number and weight; and the variation in growth for different regions of the brain is presented. (164)

WINICK, M., FISH, I. and ROSSO, P. Cellular Recovery in Rat Tissues After a Brief Period of Neonatal Malnutrition. Journal of Nutrition, 95(4):623, 1968.

Rats undernourished for the first 9 days of life and then adequately fed until weaning, did not show a reduction in cell number in various organs. This indicates that optimum feeding, begun during the period of active cell division, will correct cellular deficiencies caused by malnutrition. (165)

WINICK, M. and NOBLE, A. Cellular Response in Rats During Malnutrition at Various Ages. Journal of Nutrition, 89(3):300, 1966.

Studies on rats show that cellular effects of malnutrition depend on the growth phase at the time of malnutrition. Early malnutrition impedes cell division which is irreversible. Later malnutrition causes a reduction in cell size but can be corrected with adequate feeding. (166)

ZAMENHOF, S., VAN MARTHENS, E. and MARGOLIC, F.L. DNA (Cell Number) and Protein in Neonatal Brain: Alteration by Maternal Dietary Protein Restriction. Science, 160(3825):322, 1968.

Female rats were maintained on 8 or 27 percent protein diet by a pair-feeding schedule for 1 month before mating and throughout gestation. The brains of newborn rats from females on the 8 percent protein diet contained significantly less DNA and protein compared to the progeny of the females on the 27 percent diet. This quantitative alteration in number, as well as the qualitative one (protein per cell), may constitute a basis for the frequently reported impaired behavior of the offspring from protein deprived mothers. (167)

ZIMMERMAN, R.R. Effects of Age, Experience and Malnourishment on Object Retention in Learning Set. Perceptual Motor Skills, 28:867, 1969.

Repeated presentation of 100 6-trial problems produced significant retention of individual object discriminations in both 25-month-old and 6-month-old infant monkeys. Deprivation in the form of malnutrition of 6-month-old monkeys produced superior performance in intraproblem learning and subsequent retention, but the retention loss remained constant. (168)

Vitamin and Mineral Deficiencies

Thiamine

ALLSOP, J. and TURNER, B. Cerebellar Degeneration Associated with Chronic Alcoholism. Journal of Neurological Science, 3:238, 1966.

The neuropathological details of 7 autopsied cases are presented. The changes of Wernicke's encephalopathy were found in all but one case. The significance of this particular syndrome in relationship to atrophy of the vermis is discussed in the light of cerebellar physiology. It is concluded that chronic alcoholism may be associated with this form of cerebellar degeneration. (169)

COLLINS, G.H. An Electron Microscopic Study of Remyelination in the Brainstem of Thiamine Deficient Rats. American Journal of Pathology, 48:259, 1966.

A report on the process of remyelination in a chronic lesion in vitamin deficient rats in a restricted area in the central nervous system. The observations suggest that remyelination in the central nervous system is not restricted to a single pattern, but may be accomplished in a number of ways dependent upon the geometric features of the lesion as well as, perhaps, metabolic conditions at the time. (170)

COLLINS, G.H. Glial Cell Changes in the Brainstem of Thiamine Deficient Rats. American Journal of Pathology, 50:791, 1967.

A study of the brainstems of thiamine deficient rats reveals a series of alterations involving glial cells, which can be related to the further development of tissue breakdown. An attempt is made to correlate this data with biochemical data which suggest that oligodendrocytes, with their high transkelotase activity, would be the most probable tissue element affected by thiamine deficiency. This, however, is not conclusive. It is apparent that glial cells are affected before other tissue components. The mechanism whereby these changes may affect other parenchymal elements is discussed. (171)

FRANTZEN, E. Wernicke's Encephalopathy: 3 Cases Occurring in Connection with Severe Malnutrition. Acta Neurologica Scandinavica, 42:426, 1966.

The symptoms of Wernicke's encephalography are described and the aetiology and pathology discussed. Three case histories of patients admitted to the department of neurology with symptome of W.e. are reported. Two of the patients were chronic alcoholics, while the third had been on hunger strike for months. The results of clinical neurological investigation supplemented by electro-encephalography and air encephalography are reported. (172)

GREENHOUSE, A.H. and SCHNECK, S.A. Subacute Necrotizing Encephalomyelopathy. A Reappraisal of the Thiamine Deficiency Hypothesis. Neurology, 18:1, 1968.

Subacute necrotizing encephalomyelopathy is a disorder of early infancy with histopathologic findings which closely resemble Wernicke's disease. A defect in metabolic processes involving thiamine utilization seems a more likely etiologic explanation for this syndrome. (173)

LOPEZ, R.I. and COLLINS, G.H. Wernicke's Encephalopathy. A Complication of Chronic Hemodialysis. Archives of Neurology, 18:248, 1968.

This is a report of a patient suffering from central pontine myelinosis in association with Wernicke's encephalopathy, along with chronic renal failure. Thiamine deficiency was suspected as one of the etiological conditions in this case. Many other complicating factors were present. (174)

MANCALL, E.L. and McENTEE, W.J. Alterations of the Cerebellar Cortex in Nutritional Encephalopathy. Neurology, 15(4):303, 1965.

A case study of an 18-year-old boy, severely undernourished and having the pathological alterations of Wernicke's encephalopathy, was presented. The undernourished state was due primarily to a partial intestinal obstruction resulting from congenital duodenal bands. Since the patient was a non-alcoholic it was concluded that the cerebellar disorder was due to nutritional depletion rather than a direct toxic affect of alcohol. (175)

NEWMAN, A.J. Suspected Thiamine Deficiency in Pigs. Veterninary Record, 84:577, 1969.

Three case reviews of pigs exhibiting recumbency, irregular breathing, apparent blindness, and the inability to walk properly are given. The only consistent feature of the three cases was a movement between farms and a change of diet. Administration of thiamine brought rapid improvement. A possible explanation is that a disturbance in the metabolism of nutrients may destroy or inhibit available thiamine. (176)

NUTRITION REVIEWS, 27:54, 1969. Early Brain Stem Lesions in Thiamine Deficient Rats.

The early lesions of acute thiamine deficient rats show swelling of glial cell cytoplasm which is coincident with the onset of neurological signs. Of 68 thiamine deficient rats 37 exhibited neurological signs of deficiency, and 32 had demonstrable lesions. (177)

PENA, C.E. Wernicke's Encephalopathy. Report of Seven Cases with Severe Nerve Cell Changes in the Mamillary Bodies. American Journal of Clinical Pathology, 51:603, 1969.

Among 92 cases of Wernicke's encephalopathy received, 7 were found that exhibited unusual changes of the neurons of the mamillary bodies. Whether the changes are due to axonal injury, to transynaptic degeneration, or to a direct metabolic effect on the neurons, secondary to thiamine deficiency, still remains to be determined. (178)

ROBERTSON, D.M., WASAN, S.M., and SKINNER, D.B. Ultrastructural Features of Brain Stem Lesions of Thiamine Deficient Rats. American Journal of Pathology, 52: 1081, 1968.

The lesions of acute severe thiamine deficient rats were studied by electron microscopy. It is suggested that the failure of active transport results from interference of production of chemical energy by thiamine dependent enzymes involved in carbohydrate metabolism. (179)

TELLEZ, I. and TERRY, R.D. Fine Structures of the Early Changes in the Vestibular Nuclei of the Thiamine Deficient Rat. American Journal of Pathology, 52:777, 1968.

The effect of thiamine deficiency on the fine structure of the lateral vestibular nucleus was investigated. Material from 9 control rats and 6 thiamine deficient rats with acute central nervous system symptoms were studied with the electron microscope. (180)

YONEZAWA, T. and IWANAMI, H. An Experimental Study of Thiamine Deficiency in Nervous Tissue, Using Tissue Culture Technics. Journal of Neuropathology and Experimental Neurology, 25(3):362, 1966.

In vitro studies of thiamine deficiency in nervous tissue of rats and mice are reported. The generalized degeneration seen with high antimetabolite concentrations may result from the inhibition of all the oxidative reactions in which thiamine pyrophosphate is involved. Furthermore, the severe myelin degeneration may result from the inactivation of transketolase in the myelin-forming cells. (181)

ARAKAWA, T., MIZUNO, T., HONDA, Y., TAMURA, T., SAKAI, K., TATSUMI, S., CHIBA, F. and COURSIN, D.B. Brain Function of Infants Fed On Milk From Mothers With Low Serum Folate Levels. Tohoku Journal of Experimental Medicine, 97:391, 1969.

Brain function of exclusively breast - fed infants from 3 to 6 months old was examined by electroencephalography. Infants of mothers with low serum folacin values showed a definite delay in maturation patterns of the brain function. (182)

ARAKAWA, T., MIZUNO, T. and SAKAI, K. Electroencephalographic Frequency Patterns of Rats Treated With Aminopterin in Early Infancy. Tohoku Journal of Experimental Medicine, 97:385, 1969.

The aim of the present study is to examine the effect of folic acid deficiency upon the maturation of brain function of rats in early infancy. Folic acid deficiency was induced by injection of aminopterin to dams and offspring soon after birth. The maturation of the brain was evaluated by frequency analysis of electroencephalograms. Results showed definite differences from analytic patterns of the EEG. (183)

FLEMING, A.F. and DADA, T.O. Folic Acid and Neurological Disease. Lancet, 1(7428):97, 1966.

Seven of 15 consecutive Nigerian patients with neurologic disease had low serum folic acid activity. The serum folic acid range was low to intermediate in 12.3 percent of 228 Nigerian blood donors and in none of 50 healthy Europeans. Folic acid supplements are likely to be useful adjuncts to vitamin B preparations frequently prescribed to patients with neuropathies. (184)

GORDON, N. Folic Acid Deficiency from Anticonvulsant Therapy. Developmental Medicine and Child Neurology, 10(4):497, 1968.

Folic acid deficiency may result from treatment with various antiepileptic drugs. This seems more likely to be due to an interference with folic acid metabolism than to defective absorption. Seventy-two patients on such treatment were studied. The possible results of this deficiency are discussed with particular reference to the role of folic acid in protein synthesis. (185)

HERBERT, V. Inborn Errors in Folate Metabolism--A Cause of Mental Retardation? Annals of Internal Medicine, 68:956, 1968.

A possible link between defective folate metabolism and mental retardation is discussed. It is suggested that serum folate level determination be done on all congenital cases of mental retardation. (186)

HUNTER, R. and MATTHEWS, D.M. Mental Symptoms in Vitamin-B-12 Deficiency. Lancet, 2(7415):738, 1965.

A wide variety of psychiatric manifestations of vitamin B-12 deficiency may precede anemia or gross neurological signs by months or years. Routine screening tests for B-12 deficiency were recommended in psychiatric practice. (187)

KAUL, K.K., PRASAN, N.G. and CHOWDHRY, R.M. Further Observations on the Syndrome of Tremors in Infants. Indian Pediatrics, 1:219, 1964.

A clinical study of sixteen infants presenting with the characteristic features of the syndrome of tremors consisting of delayed development, mental retardation, skin pigmentation, anaemia and tremors, is presented. A similar clinical picture resulting from vitamin B_{12} deficiency and megaloblastic anaemia, has been reported by some workers. Our previous as well as the present observations indicate that the syndrome is not the result of vitamin B_{12} deficiency or of megaloblastic anaemia. (188)

McNICHOLL, B. and EGAN, B. Congenital Pernicious Anemia: Effects on Growth, Brain, and Absorption of B_{12}. Pediatrics, 42(1):149, 1968.

Congenital pernicious anemia from vitamin B_{12} deficiency affects growth and development. Case reports of 3 children with megaloblastic anemia at 1 year of age demonstrated a deficiency of gastric intrinsic factor. Spinal cord and cerebral lesions may also occur in the vitamin B_{12} deficiency of congenital pernicious anemia; the fact that the IQs of the patients were about 70 suggests that minor cerebral damage may have occurred. (189)

MEDICAL WORLD NEWS, 8(51):40, 1967. Lack of B-Complex Vitamin Threatens Mother and Fetus.

Although severe folic acid deficiency in pregnancy is associated with maternal megaloblastic anemia, the presence of lesser degrees of folic acid deficiency in 22 percent of 250 pregnant women indicates that this deficiency may be associated with placental abruption, miscarriage, and congenital defects. (190)

MILLER, D.R. Serum Folate Deficiency in Children Receiving Anti-Convulsant Therapy. Pediatrics, 41(3):630, 1968.

The occurrence of subnormal serum folic acid levels among 37 randomly selected epileptic children (ages 5 to 20 years) treated with anti-convulsants was 51.2 percent; slight macrocytosis was found in 18.8 percent. The etiology of decreased folate levels in patients receiving anti-convulsant medication is unknown, but the drug eficacy may depend on drug-induced folate deficiency. (191)

PANT, S.S., ASBURY, A.K. and RICHARDSON, E.P., Jr. The Myelopathy of Pernicious Anemia. A Neuropathological Reappraisal. Acta Neurologica Scandanavica, 44:1, 1968.

The neuropathological changes in 41 cases of myelopathy associated with pernicious anemia, representing 40 years of experience at the Massachusetts General Hospital were reevaluated. Observations suggest that the largest nerve fibers with the thickest myelin sheaths are most vulnerable to the deficiency state. (192)

REYNOLDS, E.H., CHANARIN, I., MILNER, G. and MATTHEWS, D.M. Anti-Convulsant Therapy, Folic Acid and Vitamin B_{12} Metabolism and Mental Symptoms. Epilepsia, 7(4):261, 1966.

Evaluation of 62 epileptic patients indicates that anticonvulsant medication has antifolate activity which in turn causes megaloblastic anemia and perhaps a variety of organic neurological disorders including mental retardation and psychiatric disorders. (193)

REYNOLDS, E.H., PREECE, J. and CHANARIN, I. Folic Acid and Anticonvulsants. Lancet, 1:1264, 1969.

There were significant differences in values for folacin in serum and cerebrospinal fluid between 60 epileptic patients treated with anticonvulsant drugs and controls. Folacin values in different mental disorders are tabulated. (194)

REYNOLDS, E.H., CHANARIN, I., and MATTHEWS, D.M. Neuropsychiatric Aspects of Anticonvulsant Megloblastic Anemia. Lancet, 1(7539): 394, 1968.

A 20-year-old girl with epilepsy controlled by phenytoin and primidone developed a drug-induced megaloblastic anemia. Successful treatment of the anemia with folic acid was associated with an increase in fit frequency and an improvement in her mental state. (195)

STONE, M., LUHBY, L., FELDMAN, R., GORDON, M. and COOPERMAN, J. Folic Acid Metabolism in Pregnancy. American Journal of Obstetrics and Gynecology, 99(5):638, 1967.

Studies of folic acid metabolism in 250 unselected pregnant women revealed that folic acid deficiency; (1) was present in 22 percent, (2) could be present without overt megaloblastic anemia, (3) increased in severity at or near term, (4) increased in complicated pregnancies, and (5) could be of significance in infant growth and development. Folic acid deficiency has been suggested as a cause of fetal brain damage, mental retardation and retarded growth and development. (196)

TORRES, I., SMITH, W.T. and OXNARD, C. Degeneration of the Peripheral and Central Nervous System in Vitamin B_{12} Deficient Monkeys. Experientia, 25:273, 1969.

This is a further study based on the findings of Oxnard and Smith, (Nature, 210, 507, 1966), regarding neurological degeneration in vitamin B_{12} deficient monkeys. There was degeneration found, and the appearance of regeneration was observed in a group of monkeys treated to reduce the B_{12} deficiency. (197)

WALKER, F.A., AGARWAL, A.B. and SINGH, R. The Importance of the Falsely Positive Reaction. Journal of Pediatrics, 75:344, 1969.

Infants with failure to thrive and who present false positive test for ketone and acetoacetic acid in the urine may have a treatable metabolic disorder. A case of a child with vitamin B_{12} deficiency is presented. (198)

WOODWARD, J.C. and NEWBERNE, P.M. The Pathogenesis of Hydrocephalus in Newborn Rats Deficient in Vitamin B_{12}. Journal of Embryology and Experimental Morphology, 17(1):117, 1967.

The hydrocephalus that is caused in fetal rats by maternal deficiency of vitamin B_{12} was not influenced by addition of X-methyl folic acid or choline chloride added to the maternal diet. (199)

ARAKAWA, T., MIZUNO, T., AND CHIBA, F. Frequency Analysis of EEG's and Latency of Photically Induced Average Evoked Responses in Children With Ariboflavinosis. Tohoku Journal of Experimental Medicine, 94:327, 1968.

Frequency analysis of electroencephalograms and estimation of the average latency of photically induced evoked responses were carried out on children aged 8 to 9 years with or without ariboflavinosis associated with low serum folate levels. (200)

BOWER, B.D. Pyridoxine, Tryptophan, and Epilepsy. Developmental Medicine and Child Neurology, 7(1): 73, 1965.

Pyridoxine deficiency (a result of an abnormal diet) has long been suspect as a cause of cryptogenic epilepsy. The history of this deficiency, the measurement of its occurrence with the tryptophan-load test, and the therapeutic application of pyridoxine were reviewed. It was concluded that the tryptophan test, as commonly used, examines only a pyridoxine-dependent pathway (an inborn error of metabolism) and that pyridoxine has not been demonstrated to be highly effective in the treatment of epilepsy. (201)

DREYFUS, P.M. Nutritional Disorders of the Nervous System. Part I--Specific Vitamin Deficiencies. Lippincott's Medical Science, 17 (3):44, 1966.

Influences of vitamin deficiencies on the nervous system are discussed in terms of various nutritional syndromes. (202)

EBERLE, E.D. and EIDUSON, S. Effect of Pyridoxine Deficiency on Aromatic L-Amino Acid Decarboxylase in the Developing Rat Liver and Brain. Journal of Neurochemistry, 15:1071, 1968.

Maternal pyridoxine deficiency begun 2 weeks before mating and continued throughout pregnancy and the nursing period resulted in diminished weight gains in the brain, the liver and the body in the first 16 days of life, as well as lowered levels of the aromatic L-amino acid decarboxylase in both brain and liver tissue. (203)

FRATTA, I., ZAK, S.B., GREENGARD, P. and SIGG, E.B. Fetal Death from Nicotinamide Deficient Diet and its Prevention by Chlorpromazine and Imipramine. Science, 145(3639):1429, 1964.

Fetal death and resorption caused by a nicotinamide deficient diet fed to pregnant rats can be prevented by administration of either chlorpromazine or imipramine. The results demonstrated that in animals fed the deficient diet the fetuses failed to survive and the nicotinamide adenine dinucleotide concentration was low; however, treatment with chlorpromazine or imipramine reversed these effects. Higher concentrations of nicotinamide adenine dinucleotide were responsible for the survival of the fetuses. (204)

FRENCH, J.H., GRUETER, B.B., DRUCKMAN, R. and O'BRIEN, D. Pyridoxine and Infantile Myoclonic Seizures. Neurology, 15(2):101, 1965.

The effects of pyridoxine on the electroencephalogram, kynureninase activity, and clinical picture of 15 children with infantile spasms were investigated. The data indicated the necessity for employing pyridoxine therapy in neonatal convulsions which have not responded to anticonvulsants. (205)

HOWELL, J.M. and THOMPSON, J.N. Observations on the Lesions in Vitamin A Deficient Adult Fowls with Particular Reference to Changes in Bone and Central Nervous System. British Journal of Experimental Pathology, 48(4): 450, 1967.

Nine cockerels and nine hens were fed hatchery diets free of vitamin A but supplemented with retinoic acid. They were made deficient in vitamin A by discontinuing the supplement and the deficiency signs were described. (206)

McKIBBIN, B. and PORTER, R.W. The Incidence of Vitamin C Deficiency in Meningomyelocele. Developmental Medicine and Child Neurology, 3:338, 1967.

Cases with meningomyelocele have a high incidence of vitamin C deficiency. Twenty-five cases with meningomyelocele were compared with 16 cases with orthopedic problems, but without neurologic diseases. The stress of repeated operative procedures in cases with meningomyelocele may deplete body stores of vitamin C. All cases with meningomyelocele should be on replacement vitamin C therapy. (207)

PILLAY, V.K. Acrocephalosyndactyly in Singapore. A Study of Five Chinese Males. Journal of Bone and Joint Surgery, 46:94, 1964.

This syndrome is noted by: high, short, broad skull; crowded teeth; syndactylism of hands and feet; mental retardation; but, no reduction in life span. Etiology: Syphilis, rickets, or other vitamin deficiencies; disturbances of endocrine function; and, injury at birth have all been attributed to this condition. (208)

SOULAYROL, R., MESDJIAN, E., LOB, H., TASSINARI, C.A. and ROGER, J. The Tryptophan Loading Test in Epileptic Children. Epilepsia, 6(4):310, 1965.

The Wachstein and Gudaitis tryptophan loading test was performed on 138 (120 mentally retarded) chronic epileptic children, 2 to 20 years of age, before and during treatment with vitamin B_6. Non-convulsive generalized epilepsy was seen especially in the vitamin-deficient group, and grand mal seizures were more frequent than tonic seizures. Follow-up studies after vitamin B_6 therapy showed a correction in the metabolic disturbance with reversal to a negative tryptophan loading test but with little, if any, effect on the clinical or electroencephalographic manifestations of epilepsy. (209)

STEWART, C.N., BHAGAVAN, H.N. and COURSIN, D.B. Effect of Biotin Deficiency on Escape and Avoidance Learning in Rats. Journal of Nutrition, 88:427, 1966.

Biotin deficient rats exhibited imparied avoidance learning. Escape learning was not affected, which suggests that the impaired avoidance learning was not due to motor impairment. Behavioral and biochemical evidence suggest that the effects of the deficiency may be on the central nervous system. (210)

EVERSON, G.J., SHRADER, R.E. and WANG, T.I. Chemical and Morphological Changes in the Brains of Copper-Deficient Guinea Pigs. Journal of Nutrition, 96:115, 1968.

The histological findings of the brain, cord, and nerve of copper-deficient guinea pigs at birth and the distribution of phospholipids in a small number of control and copper-deficient animals at birth and 63 days of age were investigated. Cerebellar folia were missing or malformed in some of the copper-deficient animals at birth and throughout the brain there was underdevelopment of myelin. (211)

EVERSON, G.J., TSAI, H.C. and WANG, T.I. Copper Deficiency in the Guinea Pig. Journal of Nutrition, 93:533, 1967.

Copper deficiency was investigated in the guinea pig because this species undergoes considerable myelination in utero. Brain abnormalities and aneurisms were observed among the progeny of female guinea pigs on a copper deficient diet. (212)

O'DELL, B.L. Trace Elements in Embryonic Development. Federation Proceedings, 27(1):199, 1968.

Deficiencies of essential minerals in the maternal diet causes damage to the developing embryo, but the nature and severity of the damage varies from element to element. A relatively narrow critical range of nutrient intake results in anomalies. The effects of cobalamin, copper, zinc and iron deficiencies are discussed. (213)

OLNEY, J.W. Brain Lesions, Obesity and Other Disturbances in Mice Treated with Monosodium Glutamate. Science, 164:719, 1969.

In newborn mice subcutaneous injections of monosodium glutamate induced neuronal necrosis on several regions of the developing brain, including the hypothalamus. (214)

O'NEAL, R.M., PLA, G.W., FOX, M.R. S., GIBSON, F.S. and FRY, B.E., Jr. Effect of Zinc Deficiency and Restricted Feeding on Protein and Ribonucleic Acid Metabolism of Rat Brain. Journal of Nutrition, 100:491, 1970.

Experiments were performed on young rats to support evidence that malnutrition during infancy inhibits maximum mental development of the individual. Tests indicate zinc is necessary for optimai synthesis of RNA and protein which is important to brain metabolism. Restricted-fed and zinc deficient rats grew very slowly, and data showed a functional deficiency of zinc in these rats even when zinc content in the brain was normal. Results show the relationship of malnutrition to metabolic processes of the brain to be associated with aspects of mental performance. (215)

ADAMSONS, K., Jr. and JOELSSON, I. The Effects of Pharmacologic Agents Upon the Fetus and Newborn. American Journal of Obstetrics and Gynecology, 96(3):437, 1966.

The most commonly used drugs in obstetric practice are reviewed. The effects of administration of agents including vitamin K, proteins and those resulting in fetal goiter are given. (216)

AMERICAN ACADEMY OF PEDIATRICS, COMMITTEE ON NUTRITION. The Relation Between Infantile Hypercalcemia and Vitamin D--Public Health Implications in North America. Pediatrics, 40(6):1050, 1967.

Excessive intake of vitamin D may produce hypercalcemia with serious sequellae. The mild form of infantile hypercalcemia, which is readily reversible with conservative therapy, consists of azotemia and failure to thrive. The more severe form, which probably has its onset in utero, includes a characteristic "elfin" facies, mental retardation, and supravalvular aortic stenosis. (217)

BARNES, A.C. The Fetal Environment: Drugs and Chemicals. IN: Barnes, A.C., ed. Intra-Uterine Development. Philadelphia: Lea and Febiger, 362, 1968.

It is difficult to establish specific causal relationships between teratogenesis and chemical agents administered to pregnant women, but it is probable that some children with defects due to chemical contact during the intrauterine period are being produced at all times. Lead, vitamin K and nicotine, among other drugs and chemicals are associated with teratongenesis in humans, and human fetal growth and development have been adversely affected by exposure to them. (218)

BRADY, J.P. and WILLIAMS, H.C. Magnesium Intoxication in a Premature Infant. Pediatrics, 40(1):100, 1967.

A mother treated with magnesium sulfate for pre-eclampsia gave birth to a premature infant with toxic effects of motor and respiratory paralysis caused by hypermagnesemia which were immediately reversed by lowering the serum magnesium level. (219)

COCHRANE, W.A. Overnutrition in Prenatal and Neonatal Life: A Problem? Canadian Medical Association Journal, 93(17):893, 1965.

Clinical and biochemical disorders occurring in the newborn which are related to the excessive ingestion of calories, fat, protein, vitamins and minerals before and after birth were outlined. Particular reference is made to the ingestion of nutritional substances during pregnancy in amounts that are relatively innocuous to the mother but may be harmful to the infant in utero. (220)

COLEMAN, E.N. Infantile Hypercalcemia and Cardiovascular Lesions. Archives of Disease in Childhood, 40(213):535, 1965.

A report concerning cardiovascular lesions in patients suffering from infantile hypercalcemia indicates that congenital endocardial fibroelastosis and the myocardial lesion of fibrocystic disease of the pancreas are related to vitamin D. (221)

FORBES, G.B., CAFARELLI, C. and MANNING, J. Vitamin D and Infantile Hypercalcemia. Pediatrics, 42:203, 1968.

Does the hypercalcemia syndrome represent a form of hypersensitivity to vitamin D? Twelve cases recently observed showed no history of excessive intake of vitamin D. A case of excessive intake of vitamin D was observed and described. (222)

KIMBROUGH, R.D. and GAINES, T.B. Effect of Organic Phosphorus Compounds and Alkylating Agents on the Rat Fetus. Archives of Environmental Health. 16(6):805, 1968.

The organic phosphorus compounds, parathion, dichlorvos, and diazinon are somewhat teratogenic in rats when given in a single dose intraperitoneally on the eleventh day of gestation. The chemicals were given in large enough dosage to cause poisoning symptoms in the dams. Decreased body weight of the fetus appears to be the most sensitive indicator of toxicity. (223)

KOCHHAR, D.M. Studies of Vitamin A-Induced Teratogenesis: Effects on Embryonic Mesenchyme and Epithelium, and on Incorporaton of H-Thymidine. Teratology, 1(3): 299, 1968.

The teratogenic action of hypervitaminosis A was studied experimentally with rat embryos, and congenital malformations associated with the excess vitamin were noted. All treated embryos were growth retarded, had abnormal dorsal curvatures, protusion of the mesencephalon, decreased somite number, and cellular disorganization. (224)

KOCHHAR, D.M., LARSSON, K.S. and BOSTROM, H. Embryonic Uptake of S35-Sulfate: Change in Level Following Treatment with Some Teratogenic Agents. Biologia Neonatorum, 12(1):41, 1968.

Effects of teratogenic procedures, namely hypervitaminosis A, trypan blue and cortisone administration, on S35 - sulfate uptake in early mouse embryos were investigated. (225)

MANIOS, S.G. and ANTENER, I. A Study of Vitamin D Metabolism in Idiopathic Hypercalcemia of Infancy. Acta Paediatrica Scandinavica, 55(6):600, 1966.

Effects are cited from tests on a markedly elevated serum vitamin D activity in 2 uniovular female siblings with a severe form of idiopathic hypercalcemia. Data suggests a genetically determined defect in the degradation of vitamin D. (226)

MEDICAL WORLD NEWS, 8(10):116, 1967. Placenta is NO Barrier to Host of Agents.

Excessive vitamin D administration in pregnant women may result in abnormalities of the major blood vessels, mental retardation, and failure of growth. Excessive vitamin K may result in hemolytic anemia and kernicterus. (227)

NORA, J.J., NORA, A.H., SOMMERVILLE, R.J., HILL, R.M. and McNAMARA, D. G. Maternal Exposure to Potential Teratogens. Journal of the American Medical Association, 202(12):1065, 1967.

In children born with anomalies it was found that each mother had been exposed to potential teratogenic agents at some time during pregnancy. No statistical difference was found between children with anomalies born to mothers exposed during the first trimester and those of mothers exposed later in pregnancy. Prescription drugs, including excessive vitamin intake, constituted the majority of exposures. (228)

PERSSON, B., TUNELL, R. and EKENGREN, K. Chronic Vitamin A Intoxication During the First Half Year of Life. Acta Paediatrica Scandinavica, 54(1):49, 1965.

A study of chronic hypervitaminosis A effects in infants under 6 months of age was reported. Noted were: (1) anorexia, (2) pronounced craniotabes and occipital edema, (3) intracranial pressure, and (4) irritability. All cases were cured relatively fast once vitamin A was discontinued. (229)

SHIRKEY, H.C. The Innocent Child. Journal of the American Medical Association, 196(5):418, 1966.

Several fetal and post-natal drug reactions are considered. Excessive vitamin K, given to a mother just prior to birth, may result in jaundice, kernicterus, mental retardation, spasticity and even death. (230)

SIMMONS, R.L. and HAWKINS, B.W. Idiopathic Hypercalcemia of Infancy. Texas State Journal of Medicine, 61(5):407, 1965.

A case of idiopathic hypercalcemia was reported along with a resume of pertinent aspects of the disease. The condition may have been caused by an inborn error of metabolism which created a hypersensitivity to small doses of vitamin D. (231)

TUNNELL, R., ALLGEN, L.G., JALLING, B. and PERSSON, B. Prophylactic Vitamin: A Dose in Sweden. Acta Paediatrica Scandinavica, 54(1): 61, 1965.

A study concerning the prophylactic dosage of vitamin A and the possible risk of hypervitaminosis A was reported. (232)

NUTRIENT METABOLISM AND MENTAL RETARDATION

CARBOHYDRATES AND PROTEIN

ANDERSON, J.M., MILNER, F.D.G. and STRICH, S.J. Effects of Neonatal Hypoglycemia on the Nervous System: A Pathological Study. Journal of Neurology, Neurosurgery, and Psychiatry, 30(4):295, 1967.

The necropsy findings of three untreated and three treated infants with hypoglycemia were compared to demonstrate that untreated hypoglycemia during the first week of life is an important cause of brain damage. The pathogenesis of brain damage from neonatal hypoglycemia is discussed. (233)

BAIG, H.A. and EDOZIEN, J.C. Carbohydrate Metabolism in Kwashiorkor. Lancet, 2(7414):662, 1965.

A study investigated the biochemical mechanism involved in the disturbance of glucose utilization in kwashiorkor. The kwashiorkor patients were found to have slightly reduced fasting blood-sugar and diminished glucose tolerance. Fasting plasma insulin was low and patients had diminished adrenocortical function. It was suggested that the disturbances in blood-glucose regulation could be explained by insulin hypersensitivity and poor pancreatic response to intravenous glucose. (234)

BLATTNER, R.J. Central Nervous System Damage and Hypoglycemia. Journal of Pediatrics, 72(6):904, 1968

Early diagnosis and treatment of congenital hypoglycemia is imperative in order to preclude the possibility of severe, irreversible brain damage resulting in mental retardation and cerebral palsy. Uncorrected and even delayed correction of neonatal hypoglycemia is often fatal. (235)

CHANTLER, C., BAUM, J.D. and NORMAN, D.A. Dextrostix in the Diagnosis of Neonatal Hypoglycemia. Lancet, 2(7531):1395, 1967.

"Dextrostix" (Ames Company) was successfully used as a screening test to detect hypoglycemia. (236)

CLINICAL PEDIATRICS, 6(2):94, 1967. Hypoglycemia in Infancy. Appraisal of the Problem, Methods of Investigation and Possible Association of Cataracts.

Three children demonstrating diverse aspects of the problem of hypoglycemia were presented. There may be a causative association between hypoglycemia and cataracts and hypoglycemia and brain damage. Hypoglycemia is found in several clinical syndromes including hypopituitarism and growth hormone deficiency, galactosemia and glycogen storage disease. (237)

COMBS, J.T., GRUNT, J.A. and BRANDT, I.K. New Syndrome of Neonatal Hypoglycemia. New England Journal of Medicine, 275(5): 236, 1966.

Clinical features and evaluations of therapy are presented on three infants with a neonatal hypoglycemia syndrome of unknown etiology. Cortisol was used with success on 1 case, and zinc glucagone and diazoxide were effective in 2 cases. (238)

COX, M. and DUNN, H.G. Idiopathic Hypoglycemia and Children of Low Birth Weight. Developmental Medicine and Child Neurology, 9(4): 430, 1967.

Of 500 infants with a birth weight below 4 1/2 pounds, 9 had idiopathic neonatal hypoglycemia, and 3 had idiopathic hypoglycemia after the neonatal period. Fourteen other children with idiopathic hypoglycemia after the neonatal period were included in the study. The outcome was significantly more abnormal in the children with hypoglycemia during or after the neonatal period than in other children of low birth weight without hypoglycemia. (239)

CREERY, R.D.G. Hypoglycemia in the Newborn: Diagnosis, Treatment, and Prognosis. Developmental Medicine and Child Neurology, 8 (6):746, 1966.

Hypoglycemia is a significant hazard to the nervous system. Serious brain damage results when it is associated with cerebral birth injury. (240)

EHRLICH, R.M. and MARTIN, J.M. Tolbutamide Tolerance Test and Plasmal-Insulin Response in Children with Idiopathic Hypoglycemia. Journal of Pediatrics, 71(4):485, 1967.

Fifteen children with idiopathic hypoglycemia were differentiated from ten normal children by means of the tolbutamide tolerance test. The insulin-glucose ratio after intravenous tolbutamide and oral glucose administration shows that children with idiopathic hypoglycemia have hyperinsulinism, which may be the important pathogenetic factor in this disorder. (241)

FISHMAN, R.A. and RASKIN, N.H. Experimental Uremic Encephalopathy. Permeability and Electrolyte Metabolism of Brain and Other Tissues. Archives of Neurology, 17 (1):10, 1967.

Acute uremia produced a generalized increase in permeability of the blood brain barrier in a radioisotope study of nephrectomized animals. This non-specific increased permeability in uremia may allow entrance of toxic compounds into the brain and thus explains the central nervous system signs of uremia. (242)

GELLER, H., CHANEY, R. and EYMAN, R. Liver Function and Serum Protein Studies on Mentally Retarded Individuals. American Journal of Mental Deficiency, 72(4):554, 1968.

This study was undertaken because of the presence of abnormal "liver function tests" in a significant number of mentally retarded patients who had been a "control group" in an investigation of hepatitis at Pacific State Hospital. It was concerned with determining the relationship of such tests to institutionalization or to biochemical abnormalities inherent in retarded individuals. (243)

HAWORTH, J.C. and McRAE, K.N. The Neurological and Developmental Effects of Neonatal Hypoglycemia: A Follow-Up of 22 Cases. Canadian Medical Association Journal, 92(16):861, 1965.

Twenty-two infants in whom hypoglycemia (blood sugar less than 20 mg./ 100 ml.) was noted during the first few days of life were followed-up when 8 to 30 months of age. This preliminary study suggests that hypoglycemia associated with neurological symptoms in the newborn period carries a poor prognosis with

respect to permanent neurological damage. Asymptomatic hypoglycemia may have a relatively good prognosis. (244)

HUNTER, A. Perinatal Events and Permanent Neurological Sequalae. New Zealand Medical Journal, 68: 108, 1968.

The preliminary results of a prospective survey of the development of infants who had been at risk from hypoxia and biochemical disturbances in the neonatal period are given. Significant neurological signs including cases of mental defect, cerebral palsy and epilepsy were found in 22.5 percent of those seen at follow up. (245)

JORDAN, W.R. The Pregnant Diabetic. Southern Medical Journal, 60(11): 1213, 1967.

A review of 105 pregnant diabetics showed that although the disease posed a problem, careful attention paid to medical and obstetrical complications improved the fetal survival rate. Problems encountered and treated included: need for increasing insulin dosage, lower renal threshold, developing acidosis, tendency towards toxemia, possible catastrophic effect of relatively minor intercurrent disease, and precipitous fall of insulin requirements at the termination of pregnancy. (246)

LANCET, 1(7538):349, 1968. Carbohydrate Metabolism in the Newborn.

Insulin secretion is low and glucose tolerance reduced during the first postnatal days. Infants predominantly affected by symptomatic hypoglycemia include: small-for-dates, premature, smaller of twins, and infants with respiratory distress or symptoms of cold exposure. In all these categories the liver glycogen is low. (247)

LONG, W.N. and HOLZMAN, G.B. Hazards to the Fetus from Maternal Diabetes. IN: Long, W.N. and Holzman, G.B., eds. Intra-Uterine Development. Philadelphia: Lea and Febiger, 427, 1968.

Respiratory distress syndrome associated with prematurity is the largest cause of death of newborns of diabetic women. Infants with diabetic mothers have problems associated with ketoacidosis, excessive size, growth retardation because of maternal vascular disease, and/or congenital malformation. (248)

MEDICAL WORLD NEWS, 8(20):37, 1967. Hormone Shot Saves Diabetics' Babies.

Treatment with epinephrine immediately after birth aids infants of diabetic mothers to avoid brain damage and respiratory distress caused by insulin build up and depressed sugar levels. (249)

NAEYE, R.L. Organ Composition in Newborn Parabiotic Twins with Speculation Regarding Neonatal Hypoglycemia. Pediatrics, 34(3): 415, 1964.

A quantitative histological examination of postmortem material taken from each member of ten pairs of monovular twins with placental transfusion syndrome was performed. There was a disproportionate reduction in the size of liver, pancreas, and fetal adrenal cortex in donor twins. Donor malnourished twins were on the arterial side whereas the recipient twins were on the venous side. (250)

NIELSEN, J. Diabetes Mellitus in Parents of Patients with Klinefelter's Syndrome. Lancet, 1 (7451):1376, 1966.

Eight (32 percent) of 25 patients with Klinefelter's syndrome were found to have near relatives with diabetes mellitus. Of the patients' parents, 8 percent of the fathers and 12 percent of the mothers had diabetes (at least 5 times the expected incidence) indicating a possible correlation between diabetes mellitus and Klinefelter's syndrome with a higher risk of sex chromosome nondisjunction in the offspring of parents with diabetes mellitus. (251)

PATERSON, P., PHILLIPS, L. and WOOD, C. Relationship Between Maternal and Fetal Blood Glucose During Labor. American Journal of Obstetrics and Gynecology, 98(7): 938, 1967.

Tests conducted with 21 pregnant women demonstrated that glucose levels of maternal and fetal blood have a close relationship during labor. The glucose concentration in the umbilical venous blood was not significantly greater than that in fetal blood. (252)

PEDERSEN, L.M., TYPSTRUP, I. and PEDERSEN, J. Congenital Malformations in Newborn Infants of Diabetic Women. Acta Paediatrica Scandinavica, Supplement 159,40, 1965.

A consecutive series of 853 newborn infants (birthweight 1,000 g. or over) of diabetic mothers, born in Rigshospitalet, Copenhagen, between January 1, 1962, and September 30, 1963, was contrasted with a control series of 1,212 infants (birthweight 1,000 g. or more) of nondiabetic mothers, born in the same departments between the autumn of 1959 and the spring 1960. Congenital malformations were found in 6.4 percent of the diabetes group and in 2.1 percent of the control group. (253)

PILDES, R.S., FORBES, A.E. and CORNBLATH, M. Studies of Carbohydrate Metabolism in the Newborn Infant. IX. Blood Glucose Levels and Hypoglycemia in Twins. Pediatrics, 40(1):69, 1967.

Carbohydrate metabolism and clinical studies on 100 pairs of twins showed a relationship between birth weight and blood sugar levels and no relationship between first or second born, or sex and blood sugar levels. The relationship between hypoglycemia and mental retardation could not be determined from this data but the possibility makes hypoglycemia a disorder that should be vigorously searched for and treated. (254)

REISNER, S.H., FORBES, A.E., and CORNBLATH, M. The Smaller of Twins and Hypoglycemia. Lancet, 1:524, 1965.

Neonatal symptomatic hypoglycemia has been presented in the smaller member of 11 sets of twins. Hypoglycemia is proposed as a possible etiological factor for the mental subnormality and slow development in the smaller twin as compared to the larger sibling. The smaller twin is thought to be an example of intra-uterine malnutrition with the larger twin as a natural control during the period of gestation. (255)

ROSENBAUM, A. and Churchill, J.A.S. Neuropsychologic Outcome of Children Whose Mothers had Proteinuria During Pregnancy. Obstetrics and Gynecology, 33:118, 1969.

A study of proteinuric mothers and the effects on offspring as to neurologic and psychologic deficits are explained. (256)

ROSENBLOOM, A.L., SMITH, D.W. and COHAN, R.C. Zinc Glucagon in Idiopathic Hypoglycemia of Infancy. American Journal of Diseases of Children, 112(2):107, 1966.

Two male infants with idiopathic hypoglycemia were successfully treated with zinc glucagon. Frequent feeding is a necessary part of management of other hypoglycemic syndromes presenting excessive insulin production or decreased glycogen release. (257)

RUSNAK, S.L. and DRISCOLL, S.G. Congenital Spinal Anomalies in Infants of Diabetic Mothers. Pediatrics, 35(6):989, 1965.

Three cases of agenesis of the lower spine in infants of mothers who had diabetes mellitus were presented. A relationship between the anomalies and maternal diabetes mellitus was suggested. The effect of insulin was considered as a possible etiologic factor. (258)

WAKOH, T., REISS, M. and HILLMAN, J. Investigation of Energy Metabolism in Mentally Retarded Patients. American Journal of Mental Deficiency, 69:319, 1964.

The investigation of basal metabolism is not easily done on mentally retarded children because of their inability to sit quietly for a sufficient period while being tested by the standard apparatus. The authors tell of an "open system" for measuring metabolism which does not require mouth pieces, etc., and consequently does not upset the children. (259)

WHITEHEAD, R.G. Biochemical Tests for Assessing Subclinical Nutritional Deficiency. Clinical Pediatrics, 6(9):516, 1967.

Possible uses, potential difficulties, and types of biochemical tests for assessing nutritional status are presented. (260)

WHITEHEAD, R.G. Biochemical Tests in Differential Diagnosis of Protein and Calorie Deficiencies. Archives of Disease in Childhood, 42(225):479, 1967.

Serum protein, the amino acid ratio, and the hydroxyproline index were used to differentiate between nutritional marasmus and two types of kwashiorkor. (261)

WILSON, J.S.P. and VALLANCE-OWEN, J. Congenital Deformities and Insulin Antagonism. Lancet, 2(7470):940, 1966.

Increased antagonism to insulin associated with plasma albumin was found in 13 of 14 mothers whose children had spinal deformities, 15 of 18 mothers whose children had upper-limb deformities, and 14 of 50 controls. (262)

BRUCK, E., ABAL, G. and ACETO, T., Jr. Therapy of Infants with Hypertonic Dehydration Due to Diarrhea. American Journal of Diseases of Children, 115(3):281, 1968.

A controlled study of 59 infants (including 5 mental retardates) with dehydration compared electrolyte solution with electrolyte free solution for rehydration. Although it takes longer for total correction of the electrolytes, a polyionic solution with 10 percent glucose is recommended for therapy of hypertonic dehydration in children. (263)

CANELAS, H.M. DeASSIS, L.M. and DeJORGE, F.B. Disorders of Magnesium Metabolism in Epilepsy. Journal of Neurology, Neuro-Surgery and Psychiatry, 28(4):378, 1965.

Eighty-three epileptic patients and thirty-four mental patients (mostly schizophrenics) were studied and their blood and cerebrospinal fluid magnesium levels compared. Hypomagnesemia was found in the interseizure period of the epileptics and the cerebrospinal fluid level was high. The cerebrospinal fluid and blood magnesium levels increased after complete crisis elicited through electroshock in the mental patients. (264)

DODGE, P.R. Some Comments on Neurological Sequelae of Diarrhea and Electrolyte Disturbances. IN: Eichenwald, H.F., ed. The Prevention of Mental Retardation Through Control of Infectious Diseases. Washington, D.C.: U.S. Government Printing Office, 285, 1968.

The neurological sequelae that result from diarrhea and electrolyte disturbances include convulsions, mental retardation, and death.Brain swelling and convulsions can be the result of hyponatremia and body fluid hypotonicity following improper fluid therapy. In a series of 53 infants with acute infectious diarrhea, seizures did not occur until the infants were rehydrated. Many of the neurological sequelae may be from improperly treated convulsions. (265)

FINBERG, L. Some Thoughts Relating to Water Movement in the Brain During Infectious Illness. IN: Eichenwald, H.F., ed. The Prevention of Mental Retardation Through Control of Infectious Diseases. Washington, D.C.: U.S. Government Printing Office, 273, 1968.

Water and electrolyte metabolism of the brain is considered in relation to treatment for central nervous system infections and the prevention of mental retardation caused by infection treatment. Specific conditions and treatment are described. (266)

FRIEDEN, E. Ceruloplasmin, a Link Between Copper and Iron Metabolism. Nutrition Reviews, 28:87, 1970.

There is now ample evidence that the copper protein of serum, ceruloplasmin, is the molecular link between copper and iron metabolism. It has been shown both in vivo and in vitro to be directly involved in iron mobilization, the rate of formation of Fe(III)-transferrin and ultimately hemoglobin biosynthesis. While the precise mechanism of this effect is not known, it appears to be directly related to the ferroxidase activity of this serum enzyme. (267)

MACAULAY, D. and WATSON, M. Hypernatremia in Infants as a Cause of Brain Damage. Archives of Disease in Childhood, 42(225): 485, 1967.

Of 122 children with hypernatremia in infancy, who were followed up 1 1/2 to 8 years after initial presentation, 100 gave no history of antecedent nervous disease. Of these, 16 were thought to have sustained brain damage, apparently from the hypernatremia. Prevention of hypernatremia is a better approach to treatment than management of the established condition. (268)

McKIBBIN, B., TOSELAND, P.A. and DUCKWORTH, T. Abnormalities in Vitamin C Metabolism in Spina Bifida. Developmental Medicine and Child Neurology, Supplement No. 15,55, 1968.

A high incidence of vitamin C unsaturation in children with meningomyelocele has already been reported. These results were obtained by the usual vitamin C saturation test, but since this investigation is not entirely reliable, confirmation was sought using an alternative investigation, namely the urinary excretion of parahydroxyphenylacetic acid. This is an intermediary in tyrosine metabolism and is excreted in excess in the absence of vitamin C. Abnormally high levels were found in 75% of the children, which corresponds with the number of abnormalities found in the earlier investigations. Possible reasons for the results are discussed. (269)

MORRIS-JONES, P.H., HOUSTON, I.B., and EVANS, R.C. Prognosis of the Neurological Complications of Acute Hypernatremia. Lancet, 2 (7531):1385, 1967.

36% of a series of 50 cases of acute hypernatremia had a neurological syndrome which included convulsions, muscular hypertonicity, and depression of consciousness. Data suggest that serum osmolality may be the most significant factor. It was suggested that, in some instances, convulsions were caused by transient cerebral edema rather than by infarction or hemorrhage, and may therefore, be preventable by improvement of the regimen of rehydration. (270)

ROSTAFINSKI, M.J. A Case of Brain Damage Due to Dehydration. Virginia Medical Monthly, 91:75, 1964.

Brain damage occurs in three main types resulting from dehydration from acute diarrhea. A case study is cited in which the patient suffered from hypertonic dehydration and it was assumed brain damage occurred from intracerebral hemorrhages. The history and literature showed that the earlier the patient suffering from diarrhea associated with acute dehydration receives medical help, the greater the chance of preventing brain damage. (271)

SEGAR, W.E. Chronic Hyperosmolality. American Journal of Diseases of Children. 112(4):318, 1966.

A 10-year-old microcephalic boy had an absence of thirst, an inability to maximally concentrate urine, and a defective osmoregulation resulting in chronic hyperosmolality. The initial treatment after 24 hours was intravenous fluid therapy. The "osmostat" abnormality may be a defect in hypothalamic secretion of antidiuretic hormone. (272)

SHIMIZU, M. Ceruloplasmin as a Controlling Factor of the Hematopoietic System. Birth Defects Original Article Series, 4(2):49, 1968.

Intramuscular injections of ceruloplasmin, folic acid, and vitamin B_{12} in combination daily for 16 weeks to rabbits produced significant increases in plasma erythropoietin as measured by erythrocytic incorporation of radioiron in rats given pooled plasma from the treated rabbits. (273)

SNODGRASS, J. Hypernatraemia and Brain Damage. Lancet, 1(7535): 186, 1968.

Brain damage from hypernatremia is described; mortality is estimated as 10-15%; and morbidity includes epilepsy and retardation. (274)

WADA, T., SAKURADA, S., OIKAWA, K., FURUKOHRI, T. and MURAMOTO, Y. Pathobiochemical Studies on Epilepsy. Daily Variation of Blood Components with Special Reference to Serum Electrolytes. Psychiatria et Neurologia Japonica, 66(8):1, 1964.

Endocrine-metabolic aspects, especially those in the serum electrolytes, were studied in both refractory and well-controlled epileptic cases. The findings may indicate some unstable pathophysiological conditions of refractory epilepsy. (275)

WADA, T., SAKURADA, S., FURUKOHRI, T., SASAKI, J. and SHIBUKI, K. Patho-biochemical Studies on Epilepsy. Part II. Diurnal Rhythm of Urinary 17-OHCS, Na, K, and Ca excretion. Psychiatria et Neurologica Japonica, 66(8):6, 1964.

Diurnal-rhythms of 17-OHCS-, Na-K- and Ca-excretion in the urine were studied in order to investigate epilepsy from the viewpoint of endocrine and electrolyte metabolism. In 17 epileptics, most of the cases showed more or less distorted excretion rhythms. The diurnal rhythm of the steroid or electrolytes seemed to be controlled by the autochthonous and endogenous rhythmic activity of the living body itself. The authors presumed that the epileptics may have a biochemical dysfunction of the central nervous system, particularly of the limbic system and the diencephalon. (276)

APPLETON, M.D., HAAB, W., CASEY, P. J., CASTELLINO, F.J., SCHORR, J. M., and MIRAGLIA, R.J. Role of Vitamin A in Gamma Globulin Biosyntheses and Uric Acid Metabolism of Mongoloids. American Journal of Mental Deficiency, 69 (3):324, 1964.

A study was made of the effect of serum vitamin A concentration on purine metabolism, and its possible relationship to the production of normal gamma globulin . It was concluded that there is a possible cause and effect relationship between low serum vitamin A levels utilized as a measure of absorption and purine metabolism, specifically with regard to the biosynthesis of gamma-globulin . (277)

CHAPMAN, M.J., DONAGHUE, E.C., SAGGERS, B.A., and STERN, J., Parotid Saliva Sodium in Down's Disease. American Journal of Mental Deficiency, 11(3):185, 1967.

Analysis of sodium concentration in parotid salivary secretions and sweat demonstrated that the content in saliva was 2 1/2 times higher in 33 cases with Down's syndrome than it was in 33 severe mental retardates of other etiology or in 17 normal adults. Inability of the salivary glands to reabsorb sodium in Down's syndrome cases may be a membrane manifestation of the generalized defect seen in trisomic cells. (278)

COBURN, S.P., LUCE, M.W., and MERTZ, E.T. Elevated Levels of Several Nitrogenous Nonprotein Metabolites in Mongoloid Blood. American Journal of Mental Deficiency, 69(6):814, 1965.

A study was made of the relative concentration of certain major nitrogenous nonprotein constituents in the blood of mongoloids. It was hypothesized that the increased level of nitrogenous nonprotein substances may be due to an increased rate of nitrogen metabolism. (279)

CULLEY, W.J., GOYAL, K., JOLLEY, D. H., and MERTZ, E.T. Caloric Intake of Children with Down's Syndrome (Mongolism). Journal of Pediatrics, 66(4):772, 1965.

A study was made of caloric intake among 23 mongoloid children with an age range of 5 to 11 years. Mean intake for boys was 1,814.0 calories and for girls, 1,555.0 calories. Final results indicated body size rather than age, was a better indicator of caloric needs, and height was a more convenient means of estimating need than weight or body surface. (280)

GRIFFITHS, A.W., and BEHRMAN, J. Dark Adaptation in Mongols. Journal of Mental Deficiency Research, 11(2):23, 1967.

Dark adaptation was found to be impaired significantly in 7 healthy mongoloid cases when compared to 7 non-mongoloid controls. A popular hypothesis is a disorder in vitamin A metabolism, influential to dark adaptation, existing in mongoloids. Several facts support this including decreased levels of serum vitamin A and analogous defects found in mongoloids and in animals deprived of vitamin A during development. (281)

McCOY, E.E., ROSTAFINSKY, M.J., and FISHBURN, C. The Concentration of Serotonin by Platelets in Down's Syndrome. Journal of Mental Deficiency Research, 12(1): 18, 1968.

Platelet and whole blood serotonin was found to be low in patients with Down's syndrome, but platelet binding of serotonin was not significantly lower which suggests that decreased serotonin synthesis accounts for the low values. It is theorized that a block of serotonin synthesis exists in mongoloids at the metabolic stage where tryptophan is hydroxylated to 5-hydroxy-tryptophan. (282)

MARGOLIS, F.J., NAGLER, R.C., and HOLKEBOER, P.E. Short-term Fluoride Excretion in Young Children. American Journal of Diseases of Children, 113(6):673, 1967.

A study of fluoride excretion following known fluoride intakes in 24 institutionalized mongoloid boys showed that a single daily dose of fluoride is retained in the body as long as multiple doses in water. (283)

MISUNSKY, A., MARKS, V., and SAMOLS, E. Insulin and Glucose Response to Glucagon in Down's Syndrome. Lancet, 2(7525):1093, 1967.

Sixteen children (5 normal, 5 mentally retarded, and 6 mongoloid) aged 20 months to 6 years, who were for 4 months on high carbohydrate diets of 1,000-2,000 calories/day, were studied for their response to glucagon. Previous data indicating that glucagon promotes insulin secretion and that such action is less effective in children than in adults were confirmed. (284)

NELSON, T.L. Milk Precipitin Disease in Mongoloids and Other Mentally Retarded Persons. Mind Over Matter, 9(2):48, 1964.

Twenty mongoloids (ages 8-45 years), closely matched with a non-mongoloid control group and a non-retarded group, were studied to determine milk precipitins. The results of the precipitin determinations indicated a significantly high incidence of precipiting and hemagglutinating antibodies to milk in the mongoloid group. (285)

NELSON, T.L. Spontaneously Occurring Milk Antibodies in Mongoloids. Journal of Diseases of Children, 108(5):494, 1964.

The similarity of the recurrent respiratory symptoms in mongoloids with the milk precipitin syndrome of Heiner was evaluated for mongoloids, non-mongoloid retardates, and normal controls. There was a discussion on possible mechanisms of the increased milk antigen stimulation among mongoloids. (286)

REISS, M., WAKOH, T., and HILLMAN, J.C. Endocrine Investigations into Mongolism. American Journal of Mental Deficiency, 70(2):204, 1965.

Data were presented on the energy metabolism of mongoloids, both waking and sleeping; on the serum PBI levels and thyroid I^{131} uptake in the resting state and after injection of TSH; on urinary 17 ketosteroids and 17-hydroxy-corticosteroids in the resting state and on the urinary 17 OCHS response to metopirone. Pre-natal endocrine environment of the mongoloid child may be instrumental to development of abnormalities in this syndrome. Chromosomal deviations during development of Down's syndrome is questioned. (287)

TECHNIQUES IN FEEDING AND THERAPEUTIC NUTRITION FOR THE MENTALLY RETARDED

ADAIR, R. Home Care and Feeding of a Mentally Retarded Child. Journal of the American Dietetic Association, 36:133, 1960.

A report is presented by a nutritionist mother on feeding techniques for the severely retarded 5-year-old child. A basic premise assumes nutritional requirements of a normal child and procedures begin at this point. Relaxed feedings give best results, and a child's wheelchair (same height as a high chair) adds convenience in feedings to both mother and child. Dietary allowances may be supplemented; for example, by ascorbic acid added to milk while the child passes through a phase of dislike for citrus fruit and juices. Feeding schedule and preparation methods are described also. (288)

AMERICAN JOURNAL OF OCCUPATIONAL THERAPY, 7(5):199, 1953. Feeding Suggestions for the Training of the Cerebral Palsied.

Difficulties encountered in feeding techniques for cerebral palsied children are handled, beginning with a program for the therapist. Awareness of a child's mental and motor capabilities is stressed and initial objectives are cautioned to be kept simple to assure success. Procedures in feeding are suggested to parents noting the importance of care and the approximation of normal conditions. Requirements for self-feeding are included, and food and utensil suggestions are detailed. (289)

ARNOLD, C.B. Feeding Suggestions. American Journal of Occupationai Therapy, 16(6):290, 1962.

Mentally retarded children with severe handicaps are described as having individual needs in the area of feeding training. Equipment such as sturdy furniture and feeding devices are detailed for use in an institutional setting. Also, for use by ambulatory and bed patients, are suggestions on the positioning of a child for feeding and other helpful feeding techniques. (290)

ARNOLD, C.B. Feeding Suggestions for the Severely Retarded Child in the Institution. American Journal of Occupational Therapy, 16:290, 1962.

The institutionalized, severely retarded child presents feeding problems which should be considered in the layout of an institution and the types of furniture and equipment chosen for it. (291)

BABCOCK, S.D. and DRAKE, M.E. A Study of the Behavioral Changes of Sixty Institutionalized Female Retardates During a Three Month Course of Treatment with Monosodium Glutamate. Training School Bulletin, 64(2):49, 1967.

Supplementing the diet of 30 institutionalized mentally retarded females with monosodium glutamate in the form of l-Glutavite appeared to benefit some states of learning and social readiness, particularly social contact. (292)

BERANT, M. and JACOBS, J.A. A "Pseudo" Battered Child. Clinical Pediatrics, 5(4):230, 1966.

The case of a 2-year, 8-month-old retarded child who was admitted to the Hadassah University Hospital in Jerusalem, Israel, for tender swelling of both legs, left shoulder and left arm was presented. From the laboratory and clinical data it was concluded that the patient was suffering from multiple nutritional deficiencies, the dominant disease being active scurvy, with severe iron and folic acid deficiencies. Treatment consisted of ascorbic acid, oral iron, multivitamin preparations, and a blood transfusion. (293)

BERMAN, H.H. and NOE, O. An Evaluation of the Nutritive Supplement Dietall. American Journal of Mental Deficiency, 62:657, 1958.

An experiment on mentally retarded children with severe feeding difficulties is described. Patients in a controlled ward environment received Dietall (precooked powdered ingredients) supplements, as did other patients having no special environmental provisions. Dietall was found reliable for fast nutrition rehabilitation, and friendly feeding procedures encouraged increased food intake and body weight gain. (294)

BERNSTEIN, N. Rehabilitating a Child with a Severe Feeding Problem. Journal of the American Dietetic Association, 36:131, 1960.

A case study of a 3 1/2 year old child with retarded growth due to insistent refusal to eat is given. The child could not relate to people and reacted by refusing all food. A program was initiated to encourage eating, and ward staff cooperated by limiting food as directed by the nutritionist who spent mealtime alone with the child. This study shows the necessity of many disciplines to accomplish goals in rehabilitation. (295)

BLANCHARD, I. Developing Motor Control for Self Feeding. Cerebral Palsy Journal, 27(5):9, 1966.

A step by step method of teaching self-feeding to cerebral palsied children is reviewed. Initially the child is spoonfed with gradual succession toward self-feeding. Frequent progress checks are viewed necessary to avoid regression. (296)

BOSLEY, E. Development of Sucking and Swallowing. Cerebral Palsy Journal, 26(6):14, 1965.

Exercise on chewing, sucking, and swallowing reflexes is considered valuable to speech development of the cerebral palsied child. Sucking is noted as important throughout the period of speech development. (297)

BOSLEY, E. Teaching the Cerebral Palsied to Chew. Cerebral Palsy Journal, 27(4):8, 1966.

This review stresses the importance of considering each part of the chewing process separately in teaching cerebral palsied children to chew. Coordinating muscles and joints is mentioned as a control for drooling and as an aid to speech. Beneficial mouth exercises are also mentioned. (298)

CHILDREN'S BUREAU PUBLICATION No. 450, U.S. Department of Health, Education and Welfare, 1967. Feeding the Child with a Handicap.

This pamphlet explains procedures for teaching self-feeding to handicapped children. It is a guide to parents to help the child learn to chew, swallow, control tongue, etc. Diagrams of modified feeding equipment are shown, menus meeting daily nutritional requirements are given, and suggestions about managing time, energy and money are stated. (299)

DANES, B.S. and BEARN, A.G. The Effects of Retinol (Vitamin-A Alcohol) on Urinary Excretion of Mucopolysaccharides in the Hurler Syndrome. Lancet, 1(7498):1029, 1967.

Daily amounts of 10,000 to 100,000 IU retinol (vitamin-A alcohol) increased urinary excretion of mucopolysaccharides in 6 cases (ages 6 months to 11 years) with Hurler's syndrome. No signs of vitamin A intoxication were seen. Increased excretion of mucopolysaccharides over prolonged periods might decrease the severity of disease in cases with Hurler's syndrome. (300)

DEKABAN, A.S. Plasma Lipids in Epileptic Children Treated with the High Fat Diet. Archives of Neurology, 15(2):177, 1966.

High fat diet treatment of 11 children (ages 1 to 6 1/2 years) with frequent and uncontrollable seizures eliminated the attacks completely in 5 and reduced them by 60-80 percent in 4. Case histories are given for 4 cases (2 with borderline intelligence and 1 with an IQ of 61). (301)

DODDS, J.M., ed. Proceedings of the Regional Workshop on Nutrition and Feeding the Handicapped. Denver: University of Colorado Printing Service, 1969.

This is a compilation of the papers presented on January 19-23, 1969 during a workshop which was sponsored by the John F. Kennedy Child Development Center, and also by the Maternal and Child Health Service, the Health Services and Mental Health Administration, the Public Health Service, and the Department of Health, Education and Welfare. The workshop's goal was to teach skills to dietitians and nutritionists to be used in diagnosing nutrition problems. Speakers included physicians, nurses, occupational therapists and a speech therapist among others. (302)

EDWARDS, M. and LILLY, R.T. Operant Conditioning: An Application to Behavioral Problems In Groups. Mental Retardation, 4(4):18, 1966.

Mealtime behavior was modified satisfactorily in a group of 26 assaultive, retarded females who were chosen to undergo operant conditioning training. (303)

ENDRES, J. and THAMAN, A. New Perspectives in Applied Nutrition for Mentally Retarded Children. Mental Retardation, 7(1):44, 1969.

A well planned feeding situation for the mentally retarded child has implications for the physical, psychological and social development of the child. The feeding program of the Child Development Day Activity Center, St. Louis University, St. Louis, Missouri, is explained as an example of the effectiveness of such a program for total development of retardates. (304)

FILIPPI, B. Antibiotics and Congenital Malformations: Evaluation of the Teratongenicity of Antibiotics. IN: Woollam, D.H.M., ed. Advances in Teratology, vol. 2. New York: Academic Press, 239, 1967.

The teratogenic effects on rat embryos of tetracycline and a penicillin-streptomycin complex both with and without vitamin replacement was studied. Vitamin supplements were used because it is known that vitamin deficiency can be caused by antibiotics and that congenital malformations appear in embryos when maternal vitamin deficits occur. (305)

GERTENRICH, R.L. A Simple Adaptable Drinking Device for Mental Retardates Lacking Arm-Hand Control. Mental Retardation, 8(3):51, 1970.

A drinking device consisting of a cup placed in a ring attached to a bar, in a manner allowing the ring to swivel freely, aided retardates in consuming liquids. The device functioned best when attached to a wheel chair, and all but the very young and most severely retarded succeeded in using the apparatus. (306)

GREENBERG, L.H. and TANAKA, K.R. Hereditary Hemolytic Anemia Due to Glucose-6-Phosphate Dehydrogenase Deficiency. American Journal of Diseases of Children, 110 (2):206, 1965.

This is a case report of an infant with hereditary hemolytic anemia. The child was maintained on folic acid treatment. Improvement in the child was said to have been due to splenectomy and oral folic acid therapy. (307)

HALL, M.E. Two Feeding Appliances. American Journal of Occupational Therapy, 5(2):52, 1951.

Two feeding appliances, used at Children's Hospital School, Baltimore, Maryland, are diagrammed and explained. Designed for cases of severe lateral immobility, both devices involve a tray which may be raised to almost mouth level. One device incorporates handles which the patient grasps to move an attached spoon in a scooping motion behind food in the tray. (308)

HENKIN, R.I. Impairment of Olfaction and of the Tastes of Sour and Bitter in Pseudohypoparathyroidism. Journal of Clinical Endocrinology and Metabolism, 28 (5):624, 1968.

Elevated thresholds for sour and bitter taste and for olfaction were found in nine mentally retarded patients (ages 13 to 26) with pseudohypothyroidism. Abnormal hard palates appeared to be responsible for the defective sense of taste. (309)

HENRIKSEN, K. and DOUGHTY, R. Decelerating Undesired Mealtime Behavior in a Group of Profoundly Retarded Boys. American Journal of Mental Deficiency, 72:40, 1967.

Four profoundly retarded young boys, considered to be the least capable in eating habits of their cottage, were placed at a special training table. Five specific forms of eating misbehavior were significantly reduced in frequency by differential movement interruption, verbal castigation, and facial disapproval. Proper eating habits were encouraged by verbal and facial approval and pats on the back. (310)

HOLSER-BUEHLER, P. The Blanchard Method of Feeding the Cerebral Palsied. American Journal of Occupational Therapy, 10(1):31, 1966.

The Blanchard spoon feeding method when used with the cerebral palsied can lead to better use of the basic reflex behavior of the lips, tongue, and throat. Such a program, as described, can result in shorter feeding time, less wasted food, elimination of coughing, choking and gagging, happier patients and more effective management by parents and personnel working with children who have feeding problems. (311)

HOUZE, M., WILSON, H.D. and GOODFELLOW, H.D.L. Treatment of Mental Deficiency with Alpha Tocopherol. American Journal of Mental Deficiency, 69(3):328, 1964.

The effect of large doses of vitamin E (alpha tocopherol) on the intellectual performance of 3 age groups of 20 mental retardates (6 patients under 10 years of age, 6 between 10 and 13, and 8 over 13 years of age) was investigated. As early as 2 weeks following treatment, some intellectual and behavioral improvement was observed by the teachers in half of their subjects. A group of 7 phenylketonurics also were treated. (312)

JOHNSON, J. and JENNINGS, R. Hypocalcemia and Cardiac Arrhythmias. American Journal of Diseases of Children, 115(3):373, 1968.

A 5 1/2-year-old mentally retarded case with rickets due to vitamin D deficiency had a complex cardiac arrhythmia which responded to oral vitamin replacement. Mental retardation and blindness created a feeding problem which in turn produced the deficiency in vitamins and calcium. Treatment with oral vitamin D (10,000 units/day) and a high calcium diet produced a normal sinus rhythm. (313)

KAUFMAN, M. Fare and Feeding for Patients with Arthritis. American Journal of Occupational Therapy, 19(5):281, 1965.

Information is provided to assist arthritic patients in activities related to food preparation and self-feeding. Prescribed diet modifications, nutritional requirements, and false information are described extensively. (314)

LONGFELLOW, L.A. Effects of Food Deprivation on Temporally Spaced Responding in Moderately Retarded Children. Dissertation Abstracts, 288(7):3075, 1968.

Food deprivation was effectively used to motivate bar pressing behavior in 3 non-verbal mental retardates. Special food providing a balanced diet was given as a reinforcer at spaced intervals. (315)

LUCCI, J.A. Daily Living Achievements of the Adult Traumatic Quadriplegic. American Journal of Occupational Therapy, 12(3): 144, 1958.

An investigation of the case records of 107 male adult traumatic quadriplegic patients was conducted to determine the achievements they had acquired in a group of selected activities of daily living, during the rehabilitation process. The eating activity necessitated assistance in food preparation and devices such as plate guards, utensil handles, and drinking tubes. This activity, although the most complex

in necessitating preparatory assistance, ranked first in the ability of the patient. (316)

LUCKEY, R.E., WATSON, C.M. and MUSICK, J.K. Aversive Conditioning as a Means of Inhibiting Vomiting and Rumination. American Journal of Mental Deficiency, 73 (1):139, 1968.

Mild electric shock was applied to a severely retarded 6-year-old boy to inhibit vomiting and ruminating. (317)

MILLICHAP, J.G., JONES, J.D. and RUDIS, B.P. Mechanism of Anticonvulsant Action of Ketogenic Diet. American Journal of Diseases of Children, 107(6):593, 1964.

Seventy-five young, female, albino mice were used to investigate the anticonvulsant effect of the ketogenic diet as compared with that of acetazolamide in children with petit mal epilepsy. The results of the mice experiment were confirmed by the clinical study which suggested that the mechanism of action of the ketogenic diet is independent of acidosis and ketosis but may be correlated with changes in the balance of electrolytes. (318)

MITCHELL, P. Buccinator Apparatus to Improve Swallowing. Physical Therapy, 47(12):1135, 1967.

A description is given of a buccinator apparatus developed to aid and improve the swallowing of some mentally retarded persons. (319)

MORRISON, D., MEJIA, B. and MILLER, D. Staff Conflicts in the Use of Operant Techniques with Autistic Children. American Journal of Orthopsychiatry, 38(4):647, 1968.

Food, used as a reinforcer in operant conditioning of a child, caused conflicts among personnel. Some viewed this experiment as scientific methodology, others as a "last resort". Ward and research staff found themselves acting emotionally rather than rationally, and the need for communication to solve difficulties was stressed. (320)

PITTS, J.L., WHITE, B.D. and COATES, M.L. An Approach to Meeting the Nutritional Needs Among Tube-Fed Mentally Deficient Children. American Journal of Mental Deficiency, 65:489, 1961.

Sustagen, a powdered, whole food preparation mixed with water, was given to a group of severely retarded children instead of their previous multiple daily gavages. Administered through an indwelling catheter the approach alleviated problems and lessened dangers associated with multiple gavage feedings by non-professional personnel. No complications emerged and general conditions of the group improved. (321)

POWELL, M. An Analysis of Behaviors to Promote Independent Feeding Skills. IN: Nursing in Mental Retardation Programs. Children's Bureau: 4th National Workshop for Nurses in Mental Retardation, 89, 1967.

Self-feeding must be programmed by the nurse, in its simplest form, for the mental retardate. Handling the child learning self-feeding involves: observation, interviewing, identification of the problem, planning, intervention and evaluation. Familiarity with family and the child's history is needed in addition to repetition and reinforcement when working with the mentally retarded child. (322)

REGER, R. and DAWSON, A. Useful Adaptation Devices for Quadriplegics. American Journal of Occupational Therapy, 15(5):203, 1961.

A metal band with spoon or fork attached is pictured and explained. The device makes it possible for a patient with flaccid paralysis of the hand to feed himself. (323)

SNOW, R., SACKS, M.O. and CORNBLATH, M. Ketotic Hypoglycemia in a Russell Dwarf. Journal of Pediatrics, 69(1):121, 1966.

A Russell dwarf with recurrent hypoglycemic seizures was found to have ketotic hypoglycemia. The patient had a reduction of seizures on a regimen of low fat, high carbohydrate diet, and anticonvulsants. (324)

SPRADLIN, J.E. The Premack Hypothesis and Self-Feeding by Profoundly Retarded Children. Parsons State Hospital and Training Center, Parsons, Kansas, 1964. (A case report)

A profoundly retarded preadolescent girl is taught self-feeding with a spoon. Training steps are outlined. (325)

WALKER, G.H. Nutrition in Mentally Deficient Children. Journal of The American Dietetic Association, 31:494, 1955.

Special diets and feeding methods are frequently necessary for the mentally retarded child; especially for the child with cerebral palsy, the mongoloid, the epileptic, and the cretin. (326)

WHITE, J.C. and TAYLOR, D.J. Noxious Conditioning as a Treatment for Rumination. Mental Retardation, 5(1):30, 1967.

Electric shock was administered to two ruminators with subsequent decrease in the behavior. The usefulness of shock in treating such behaviors is discussed. (327)

WHITNEY, L.R. Behavioristic Nursing. 90th Annual Meeting of the American Association on Mental Deficiency, Chicago, Illinois, 18, 1966. (Paper.)

Strengthening and weakening the behaviors of retardates to achieve independent skills in daily living, as a nursing responsibility, is discussed. Behavior is defined, and changes are induced by changes in environmental conditions. A procedure for strengthening and maintaining self-feeding skills is mentioned, and techniques of shaping and fading are described. (328)

WHITNEY, L. Operant Learning Theory: A Framework Deserving Nursing Investigation. Nursing Research, 15(3):229, 1966.

This is a review of literature on operant learning and its implications to nursing. An explanation of terms is given as well as mention of the situations in which operant learning deserves nursing attention. Operant learning and the mentally retarded child is stressed. (329)

WHITNEY, L.R. and BARNARD, K.E. Implications of Operant Learning Theory for Nursing Care of the Retarded Child. Mental Retardation, 4(3):26, 1966.

Effective operant conditioning of a mental retardate to spoon-feed herself is described. Positive and negative reinforcement was used. Once spoon-feeding was learned, systematic reinforcement was withdrawn and extinction occurred. Relearning progressed rapidly and training of counselors was undertaken to avoid extinguishing in examiner's absence. (330)

WOLFSON, I.N., FLACK, K.E. and WEST, L. Special Diet for Feeding Advanced Spastics and Low Grade Mental Defectives who Present Feeding Problems. American Journal of Mental Deficiency, 58:465, 1954.

The feeding of severe spastic patients is aided by a powdered diet formula, Dietall, that is mixed with water and served as a cereal. Nutritive value proved satisfactory in patients given this formula during a two year experimental program. General physical conditions of patients improved; they received easily controlled nourishment; and Dietall is economical financially, as well as time and labor saving, in the preparation and serving of food. Advanced patients rejected the formula and associated it with low grade patients. (331)

ZICKEFOOSE, M. Feeding the Child with a Cleft Palate. Journal of the American Dietetic Association, 36:129, 1960.

A study of feeding problems of children with a cleft palate revealed that most of them, with the aid of parental ingenuity, have little serious difficulty in feeding. Food intake patterns of these children are often similar to those of other children. Suggestions about food preparation and feeding techniques are also explained. (332)

ZIMMERMAN, M.E. Analysis of Adapted Equipment. American Journal of Occupational Therapy, 11(4):229, 1957.

Stress is placed on knowing the anatomy and kinesiology of body parts before one can assess feeding suggestions for mentally retarded and handicapped patients. An explanation of basic body motions, in addition to an analysis of materials best suited to feeding device construction, is provided. A table gives evaluations of the biodynamics of eating motions, methods and devices used, equipment, and types of food most suitable for handicapped patients. (333)